THE DISEASES OF THE HEART AND OF THE AORTA.

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AND OF

THE AORTA.

BY

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EIGHTY-ONE ILLUSTRATIONS.

PART I.

"Vaga enim experientia, et se tantum sequens, mera palpatio est. . . At cum experientia lege certa procedet, seriatim et continenter, de scientiis aliquid melius sperari poterit."—*Novum Organum.*
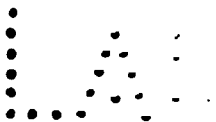
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TO

WILLIAM STOKES, M.D., F.R.S., D.C.L. Oxon.,

LL.D. Edin. and Cantab.;

REGIUS PROFESSOR OF PHYSIC IN THE UNIVERSITY OF DUBLIN;

PHYSICIAN TO THE QUEEN;

PRESIDENT OF THE ROYAL IRISH ACADEMY; ETC., ETC.

AS A TRIBUTE OF ADMIRATION FOR HIS EXALTED CHARACTER AND PRE-EMINENT

ATTAINMENTS IN MEDICINE,

AND AS A TOKEN OF GRATITUDE FOR THE MANY AND GREAT FAVOURS

RECEIVED FROM HIM THROUGH A PERIOD OF MORE THAN TWENTY YEARS,

THIS BOOK IS INSCRIBED,

BY HIS FRIEND AND FORMER PUPIL,

THE AUTHOR.

PREFACE.

AT the outset it may be asked, why add another to the long list of treatises on the Diseases of the Heart already extant? To this question I would answer, firstly, because I think the subject needs revision in the light of modern pathology; and secondly, because I believe that, as the result of long and earnest clinical study, I can contribute something towards placing this department of medicine upon a broader and firmer basis than that which it has hitherto occupied.

In undertaking a task of such magnitude, I was sensible of the great and numerous intrinsic difficulties which it involved, and no less conscious of the many defects inherent in myself, by which these difficulties were in no small degree enhanced. And now that the work, sketched in outline nine years ago, lies before me with all its imperfections, I am the less satisfied with my performance, because of the ideal standard of excellence at which I then aimed. For all such, I would solicit the considerate indulgence of my professional brethren; especially of the Dublin School, to which I have the high honour to belong.

To the great masters, Corvisart, Laennec, Bertin, Bouillaud, Hope, Walshe, Stokes, and Corrigan, students of Cardiac Disease owe much. An acknowledgment is likewise due to many others, by whose labours our knowledge of this subject has been advanced. These eminent observers have not only laid the foundation of accurate knowledge in the domain of cardiac pathology, and constructed its rudiments out of the crude materials strewed around them by the labours of their predecessors, but have de-

veloped into maturity the infant science which they created. Despite the speculative physiology of the period, and the paucity of instrumental appliances then available for the purposes of physical exploration, in less than a quarter of a century from the date of Laennec's great work on *Mediate Auscultation*, the diagnosis of cardiac disease had attained to the proportions of a science. The present advanced state of knowledge of the physiology of the heart, due to the recent experimental researches of Chauveau and Faivre, E. Weber, Ludwig, the brothers Cyon, and, especially, those of Marey and his followers, has prepared the way for a more precise cardiac pathology. In no department of medicine is a full and exact knowledge of the normal function of the organs under examination of greater importance, than in that which embraces the study of Diseases of the Heart and Great Vessels. Without such knowledge, a rational system of diagnosis and treatment is impossible.

If an example were demanded of the direct application of physiology to the study of disease, no more forcible illustration of such could, in my opinion, be adduced, than that supplied by the heart. The entire cycle of cardiac phenomena in the state of health must be thoroughly apprehended, else a correct interpretation of the derangements exhibited in the various forms of disease to which the heart is subject, cannot be attained ; and this, not only in regard to the rhythm and duration of these phenomena, but also in relation to their causes and associations, whether simple or multiple. Up to a very recent period, the knowledge available in this department of physiology was inadequate for the purpose of accurate clinical research ; hence, the want of precision in regard to differential diagnosis which characterizes the works of many writers on Diseases of the Heart. The great mass of well observed facts, however, which they have collected ; the correct, though broad lines of distinction they have drawn between the different genera

of Cardiac Disease; the extensive knowledge of etiology and prevention, and the prescience in regard to results, shadowed forth in their writings, have contributed to the advancement of this important subject, in a degree scarcely appreciable by those who now engage in the same line of study.

It is no disparagement, therefore, to the labours of these distinguished men, and no slight upon their eminent services in the cause of medical science, to assert that their works require revision in order to bring them up to the standard of present knowledge. The recent edition (the fourth) of Dr. Walshe's admirable treatise, issued while this work was in progress, contains much of the information which it had been my ambition to supply. I venture to hope, nevertheless, that in so extensive a field, my labours may not be entirely unproductive of useful results.

This volume has far exceeded the limits originally designed. If an apology be needed, I can only plead, that a curtailment of its proportions, while it would have spared me much labour, would have been at the expense of an inadequate treatment of the subject.

As physician to a great hospital for the last thirteen years, I have enjoyed many advantages for the prosecution of my favourite study The liberal and enlightened spirit in which the Mater Misericordiæ Hospital has been founded and conducted by the excellent Sisters of Mercy; the admirable economic and general arrangements of the Institution ; and the unfettered liberty to admit patients conceded to the Medical Officers, combine to insure a supply of cases amply illustrative of any disease, to which, for the time, special attention may be directed.

For assistance rendered me in the preparation of this work, I am indebted to numerous kind friends. To the several gentlemen, now dispersed through many countries or settled in prac-

tice in their own, who acted as my clinical clerks, and assisted me in observing and noting the cases recorded in the text, and in applying the crucial test of diagnosis in the *post mortem* room, this book may, perhaps, serve as a not unpleasing memorial of our conjoint labour and common study. To my hospital colleagues, for the privilege, so kindly granted me, of free access to the patients under their charge; and, in an especial manner, to Dr. Nixon, for the many admirable sphygmograms dispersed through the succeeding pages with which he has supplied me, I beg to express my deep sense of obligation. For the great and valuable assistance rendered by my friend Dr. George F. Duffey, in revising the sheets (from page 225), and pointing out references, and especially for the preparation of the copious Index, which has been entirely executed by him, I hereby tender my warm and sincere thanks. The woodcuts have been executed by Mr. George A. Hanlon.

30, Harcourt-street, Dublin.
July, 1875.

CONTENTS.

CORRIGENDA.

Page 60, last line, *for* Achiv., *read* Archiv.
,, 70, line 29, *for* diagramatic, *read* diagrammatic.
,, 78, ,, 8, *for* dilitation, *read* dilatation.
,, 91, ,, 25, *for* Chaveau, *read* Chauveau.
,, 96, last line, for *Hospitaux*, read *Hôpitaux*.
,, 106, ,, 31, *for* diagonosis, *read* diagnosis.
,, 108, ,, 2, *for* æriform, *read* aeriform.
,, 112, ,, 17, *for* diognosis, *read* diagnosis.
,, 115, ,, 37, *for* shoc, *read* shock.
,, 123, ,, 4, *for* interrupted, *read* continuous,
,, 129, 162, and 163, *for* Guttemann, *read* Guttmann.
,, 129, ,, 16, *for* Giegel, *read* Geigel.
,, 145, ,, 38, *for* shoc, *read* shock.
,, 739, ,, 17, *for* cysticeri, *read* cysticerci.
,, 804, ,, 27, *for* sitting, *read* recumbent.
,, 842, ,, 24, *for* M. Guizot, *read* M. Guyot.

THE HEART.—(*Frontispiece.*)
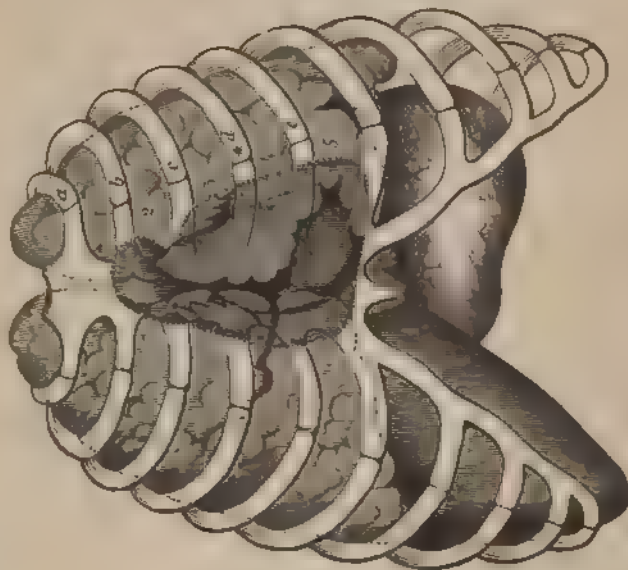
FIG. A.

Showing the relative position of the heart, lungs, stomach, and liver; also
the outline and extent of the regions of superficial and deep precordial dulness.

FIG. B.

Showing the outline of the heart, and the position of its several parts in
relation to the walls of the chest and the nipple.

The letters indicate the ribs and costal cartilages; the numerals indicate the curve upon lung intercostal spaces; the asterisk indicates the left nipple.
For description see page 1. The figures have been modified from Flint.

THE DISEASES

OF

THE HEART AND THE AORTA.

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE HEART.

THE heart, the centre of the circulatory system, occupies the lower part of the mediastinum, and the anterior inferior and internal portion of the left side of the thorax; it is enclosed in a twofold membranous sac, the pericardium, and interposed between the pleuræ, which, in close juxta-position by their outer surfaces anteriorly and posteriorly, forming the lateral boundaries of the corresponding portions of the mediastinum, are divaricated in the intermediate space to receive the heart and pericardium.

The region in front of the heart is named the *precordium*, and that portion of it in which the pericardium is not covered by the edges of the lungs, the *superficial precordial region.* This last-mentioned space, in which the pericardium comes into contact with the anterior wall of the chest, is quadrilateral in figure as correctly described by Sibson.* It measures about three inches in its transverse, and two inches in its vertical diameter; bounded superiorly by a horizontal line corresponding to the upper edge of the fifth costal cartilage, and extending from the middle line of the sternum to a point within an inch and a-half of the left nipple line; below, by a line on a level with the upper edge of the sixth costal cartilages; on the right side, by the mid line of the sternum; and on the left, by a curved line, convex outwards, commencing one and a-half inch inside the nipple line at the upper edge of the fifth costal cartilage, and terminating at the junction of the sixth and seventh costal cartilages near the sternum, two inches internal to the nipple line. These boundary lines are constituted on three sides by the edges of the lungs, viz., on the right side by the edge of the right lung, and superiorly and

* *Medical Anatomy.*

externally by that of the left lung. The inferior boundary is formed by the diaphragm. This space corresponds to the area of absolute precordial dullness, but varies much in figure and dimensions in different individuals, being less extensive in the robust, and occasionally all but abolished in the subjects of pulmonary emphysema. The dimensions given are those presented at the acme of inspiration; but during expiration they are considerably extended, owing to retrocession of the edges of the lungs.

Gendrin * says the position of the edge of the left lung may be marked out by a line drawn from the junction of the cartilage of the second rib with the sternum, downwards and outwards to the extremity of the last rib; and that of the right lung by a line carried from the upper extremity of the mesial line of the sternum, downwards and outwards to the anterior extremity of the first right false rib. In the dead body they do not advance so far as in the living.

The space known as the *deep precordial region,* or that within which modified cardiac dulness is detectible during life, corresponds in figure and dimensions to the anterior surface of the heart. It is irregularly triangular in outline, the vertex above formed by the transverse portion of the arch of the aorta, and extending from the upper edge of the second costal cartilage of the left side at its junction with the sternum, to the lower edge of the corresponding portion of that of the right side. The vertex is rounded, and about two and a-half inches in diameter. The base is below, and corresponds to a horizontal line about four inches in length, connecting the lower edges of the sixth costal cartilages of the opposite sides. The left boundary, slightly convex outwards, extends between the upper edge of the second costal cartilage of that side, half an inch external to its union with the sternum, and the upper edge of the sixth of the same side, one inch inside the nipple line. The right boundary corresponds approximately to a line carried from the lower extremity of the right second chondro-sternal articulation, vertically downwards to the upper extremity of that of the sixth. This region includes within its boundaries that known as the superficial precordial region, and yields, to the extent of the latter, *absolute* percussion dullness, but elsewhere *qualified* and barely perceptible

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* *Leçons sur les Maladies du Cœur et des Grosses Artères,* tom. i., 1841–42.

dullness, which, when the edges of the lungs are inflated, is converted into absolute resonance.

The pericardium consists of a fibrous and serous layer, the former, external, conical in figure, the base resting on the central leaflet of the cordiform tendon of the diaphragm, with which in the adult it is intimately incorporated, presents nine funnel-shaped openings for the passage of the great vessels which enter and leave the heart, viz., the aorta, the two primary branches of the pulmonary artery, the superior and the inferior vena cava, and the four pulmonary veins. In the fœtus there is an additional aperture for the ductus arteriosus, but after the closure and atrophy of this vessel, that opening becomes likewise obliterated. The edges of these several openings are everted, and incorporated with the outer coats of the vessels, with the single exception of the ascending vena cava, which pierces the diaphragm and the fibrous pericardium sharply, and at once enters the right auricle. This outer layer of the pericardium consists of fibrous tissue, chiefly of the white or inelastic kind, the fasciculi of which mutually intersect at all conceivable angles; there is likewise a large proportion of yellow elastic tissue, consisting of curling and branching filaments, varying in diameter from 1-10,000th to 1-6,000th part of an inch, and unaffected by strong acetic acid.

The serous layer of the pericardium, after lining the fibrous envelope, is reflected thence upon the great vessels at the base, to the heart, which it completely invests, constituting the *epicardium;* it forms a common envelope for the aorta and the pulmonary artery, about two inches of which are within the pericardium; the portions of the vessels to this extent, which are in mutual contact, being alone uncovered by serous membrane. From the root of the aorta and pulmonary artery the serous envelope is reflected upwards over the anterior and superior surfaces of both auricles, to the bifurcation of the pulmonary artery, whence it is reflected forwards upon the trunk of that vessel, and by it conducted downwards to the ventricles, expanding in its course into the common envelope for the aorta and pulmonary artery, already described. To the right it is reflected over the descending cava, covering it on the anterior half of its circumference to the extent of one inch of its length; it thence

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passes downwards in front of the right pulmonary veins, to the
ascending vena cava, which, to the extent of half an inch of its
length, it invests anteriorly and laterally. Between the two
cavæ, and in front of the right pulmonary veins, the serous mem-
brane dips into an oblong fossa, the long diameter of which is
vertical, and the depth of which is increased by tension of the
venæ cavæ. From the roots of the great vessels at the base it
descends over the anterior surface of the heart, to the inferior
margin and apex, whence it is reflected over the inferior surface,
and upwards over the posterior surface, investing in the last-
mentioned portion of its course the posterior surface of the left
ventricle and auricle; and from the upper margin of the latter,
ascends to the bifurcation of the pulmonary artery, where, on a
level with the anterior reflection previously described, it is re-
flected backwards to the posterior wall of the pericardium, and
thus descends to the diaphragm. On the right and left side it
invests the posterior surface of the pulmonary veins at their
termination, to the extent of one inch of their length, being also
inflected forwards between them to a variable extent. In some
subjects this inflection does not exist. The lower surface of the
inferior pulmonary vein, on both sides, is completely invested
by serous membrane, whilst the superior surface is, to an equal
extent, uncovered. The anterior surfaces of these vessels are
covered only to the extent of about half an inch.

It follows, therefore, that the sac of the pericardium is divided
by the reflection of its serous lining into two great pouches or
recesses, viz. : one, by much the larger, in front of the heart, and
separated at its circumference from the other by a double sep-
tum, formed by the reflection, *dos à dos*, of the two layers of
serous membrane already described. This septum, on the right
and left sides, includes the corresponding pulmonary veins, whilst
superiorly its constituent laminæ, extended somewhat tensely
between the superior posterior margin of the auricles inferiorly,
and the bifurcation and both lateral branches of the pulmonary
artery superiorly, are separated only by some dense areolar
tissue. The anterior recess is traversed superiorly by the trunks
of the aorta and pulmonary artery, which, through the greater
portion of their course within the pericardium, occupy a common

tubular passage between the two layers of serous membrane, but near their point of exit from it acquire each a proper serous investment.

The venæ cavæ, and both branches of the pulmonary artery, may be regarded as outside the pericardium ; projecting, however, to an unequal extent of their circumference into its cavity, the former in the posterior, and the latter in the superior wall.

The subserous lamina is of variable thickness and density in different portions of its extent; thus, beneath the fibrous pericardium it is represented by the deepest portion of that membrane, which is somewhat less dense in structure than the remainder ; in this stratum the minute vessels and capillaries of the pericardium are distributed ; on the great vessels it is more lax, admitting of easy detachment of the serous layer; whilst upon the heart it forms a remarkably dense but thin lamina, the *fascia cordis*, which is inseparably united with the basement membrane and epithelium, upon the one hand, and with the sparse perimysium or connective tissue of the heart, upon the other. In the course of the cardiac grooves only, where the principal blood vessels and nerves with a variable quantity of fat are lodged in its meshes, is this tissue sufficiently lax to admit of easy separation of the serous investment from the heart. In the interspaces between the grooves and over the surface of the heart generally, imbedded in the subserous lamina, the lymphatic vessels and the primary branches of distribution of the cardiac nerves are spread out. This tissue is likewise the seat of the "*white*," or "*milk-spots.*" There is usually but one such, which is situated upon the anterior surface of the right ventricle, irregularly circular, and varying in size from a fourpenny to a half-crown piece. When additional spots are present, they are found upon the anterior surface of the apex and that of the right auricular appendix. These latter are never met with in the absence of the former, which, on the contrary, exist in greater or less perfection on the hearts of most adult and aged persons, unaccompanied by similar appearances on other portions of the surface of the heart. Such spots were formerly, but incorrectly, regarded as the result of limited pericarditis. I have repeatedly examined them with much care, but never found

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fibrinous exudation upon the epithelial surface, which here, as upon other portions of the surface of the heart, has invariably, in my experience, presented the polished and glistening appearances characteristic of its healthy condition. They arise, manifestly, from chronic irritation of the superficial surface of the heart, the result of excessive and long continued friction, to whatever cause due. Persons long engaged in some bodily labour, involving great muscular strain and habitual distention of the right chambers of the heart, are most likely to exhibit typical " milk spots." Hence, their usual position on the anterior surface of the right side of the heart, opposite the unyielding surface presented by the sternum.

Examined microscopically, the subserous lamina of the pericardium is found to consist mainly of white fibrous tissue, the fasciculi of which overlie and interlace with one another at all conceivable angles. It includes, also, a large proportion of yellow elastic tissue, composed of fine curling filaments of an average diameter of 1-17,000th part of an inch. This latter tissue confers upon the pericardium in an eminent degree the property of elasticity, in virtue of which it is capable of accommodating itself to variations in volume of the several chambers of the heart, during contraction and relaxation respectively. The capacity of the pericardium is accurately adapted to the volume of the heart at all periods of cardiac action. Its walls are therefore constantly in a state of tension during the continuance of cardiac movement, no less during the systole than the diastole of the ventricles.

This accuracy of adaptation of the pericardium is, no doubt, mainly due to the constancy of volume of the heart at all periods of its action, a circumstance to be attributed to the alternating contraction and relative capacity of the auricles and ventricles, and of the roots of the great vessels connected with them, and included within the pericardium. If the chest of a still living animal be laid open, it will be found impossible to pinch up the pericardium between the finger and thumb, and difficult even by means of a pair of good forceps, as long as the heart continues to pulsate.

The purposes of the pericardium seem to be to insulate the

heart, and by presenting to it a smooth and moist surface, and excluding adjacent organs, to insure its free and unimpeded movement in the performance of its function.*

There is yet another purpose served by the pericardium, to which attention has not been hitherto directed, namely, that of protecting the heart from mediate atmospheric pressure at these periods of its action, when, owing to asynchronism between its movements and those of the lungs and thorax, the circulation would be liable to obstruction in the more thin-walled of its chambers, and in the roots of the great vessels, from pressure of the expanding lungs or receding chest walls.

The pericardium is firmly attached, in front, to the sternum, through the anterior mediastinum; posteriorly, to the vertebral column through the posterior mediastinum; superiorly, to the first ribs and clavicles through the reflections of the cervical fascia upon the great vessels connected with the aorta and descending cava, which are themselves incorporated by their external coats with the fibrous lamina of the pericardium where they respectively penetrate it, and with the serous layer where the latter is reflected upon them. The phrenic nerves likewise connect the pericardium superiorly with the cervical vertebræ, and their arterial *comites* with the subclavian arteries through the medium of the internal mammaries.

Inferiorly the pericardium is fixed and held tense by its attachment to the great phrenic centre of the diaphragm; and by the tension of the pleura reflected from it upon the lung on either side, it is subjected to constant, considerable, and in the normal state, equal traction laterally.

It is the derangement of balance between the tractile forces thus exercised upon the two sides of the pericardium, owing to collapse of either lung, which determines its lateral displacement to the opposite side in cases of copious aeriform

* Senac (*Traite de la Structure du Cœur, de son Action, et de ses Maladies*, 1749) says the uses of the pericardium are to facilitate the movements of the heart, by presenting opposed moist surfaces, the halitus being confined and thereby preserved, and to fix the roots of the great vessels, and maintain unalterable relationship between them and the heart, and thus favour the entrance and exit of the blood.

or liquid effusion into one side of the chest, according to the experiments of Dr. R. Douglas Powell,* to which I shall have occasion to revert.

Between these several fixed points of attachment the pericardium is kept in a state of tension sufficient to neutralize the pressure of the expanding lungs, and to diminish, at least, that of the receding sternum. Were it otherwise, the blood would be inevitably forced back from the auricles upon the great veins, as often as auricular diastole should happen to coincide with the acme of inspiration, during which the anterior edges of the lungs advance between the pericardium and the sternum with a force equivalent to several inches of mercury; and a like inconvenience, but in a much less degree, would probably arise from the retrocession of the sternum at the acme of expiration, were it not for the protection afforded to the subjacent auricles by the tense intervening wall of the pericardium.

From what has preceded, it follows that the pericardium is endowed, in an eminent degree, with the property of resistance to extension. To this property, conjoined with the strict adaptation of its capacity to the volume of the heart, must be attributed the formidable train of symptoms, of a syncopal character, which follows from rapid effusion, of whatever kind, into the sac of the pericardium.

In such a case the effused liquid is interposed between a tense and unyielding membrane, and an organ whose chambers alternately contract and expand. Now, inasmuch as the force of expansion of any of the chambers of the heart is the equivalent of the blood-pressure expended upon its internal surface during its relaxation, it is manifest that this pressure must be sufficient, not only to divaricate the walls upon which it is exercised, but likewise to overcome the resistance of the pericardium, else the expansion of the chamber will be limited by the effusion, and the volume of blood circulating through it diminished in the same proportion. Hence, the feeble impulse of the heart, the weak and flickering pulse, and the pallid and congested surface in cases of moderate but acute effusion into the pericardium, and

* *Medical Times and Gazette,* 30th January, 21st August, 1869, and *British Medical Journal,* 17th July, 1869.

the mortal syncope that not unfrequently ensues when the effusion has been rapid and copious.

Sibson says that twelve to eighteen ounces of liquid may be injected into the healthy pericardium. This result is due, in part, to forced expansion of the sac, but mainly to evacuation of the chambers of the heart. In chronic pericarditis as much as three pints of serous effusion have been found in the pericardium, the structure of the sac having undergone slow and general extension.

If the pericardium be laid open whilst the body is still warm, its serous surface will be found moistened throughout by a fine halitus, which, coincidently with the cooling of the body, becomes condensed into a clear serum, amounting in quantity to a few minims. The surface whence this moisture is exhaled is smooth, glistening, and transparent, and coated with a single layer of nucleated pavement epithelium, the particles of which are oval in shape, and of an average long diameter of 1-1100th part of an inch, and a short diameter of 1-1700th.

This surface, when the subject of inflammation, loses its smoothness and transparency, and becomes studded with effused lymph, affecting a flocculent or reticular disposition, whence arises the phenomenon of *frottement* or friction-sound during the movements of the heart. The heart is now likewise brought into view, the portions of the organ visible from the front being the greater part of the right ventricle, a part of the left ventricle including the apex, the appendices of both auricles, and a part also of the sinus of the right auricle. The intra-pericardial portions of the pulmonary artery, aorta, and superior vena cava are likewise exposed to view, as are the trunks and primary branches of the two coronary arteries occupying the anterior cardiac grooves.

The heart is irregularly triangular, and furnishes by its figure a standard of comparison, bodies shaped like it being described as *cordiform.* It presents three surfaces, of which the posterior is convex, resting superiorly on the posterior mediastinum and in immediate contact with the œsophagus, and inferiorly in a deep fossa on the internal surface of the left lung. This surface is formed entirely by the left auricle and ventricle, and presents the auriculo-ventricular groove, containing a branch of the ante-

rior coronary artery, a branch of the coronary vein, and numerous
branches of the anterior coronary plexus of nerves. The inferior
surface is flat and horizontal, formed in five-sixths of its extent
by the ventricles, and in the remaining sixth by the right auricle;
of the ventricular portion of this surface the anterior third is form-
ed by the right ventricle, and the posterior two-thirds by the left
ventricle. It presents the continuation of the ariculo-ventricu-
lar, and the so-called posterior interventricular groove; the former
containing the continued trunk of the posterior coronary artery,
and an anastomosing branch from the anterior coronary, together
with branches of the cardiac nerves, and the great coronary vein;
and the latter, the terminal branch of the posterior coronary
artery, branches of the cardiac nerves, and a large tributary of
the coronary vein. This surface rests upon the central leaflet of
the cordiform tendon of the diaphragm, and to a slight extent
upon the muscular portion between the central and left leaflets.
The anterior surface of the heart, irregularly triangular in out-
line, and slightly convex, presents to view the anterior interven-
tricular, and the auriculo-ventricular grooves, in which are lodged
the coronary arteries and nerves; likewise. the roots of the pul-
monary artery and aorta overlapped by the auricular appendices.
It is formed in the basic sixth of its extent by the auricles, chiefly
the right, and in five-sixths by the ventricles, the right ventricle
constituting two-thirds, and the left only one third of this latter
portion. It is related, in the superficial precordial region, to the
lower portion of the sternum left of the middle line, the left
costal cartilages from the middle of the fourth to the upper
margin of the sixth, and the corresponding intercostal spaces to
one inch inside the nipple, and to the left triangularis sterni
muscle and internal mammary vessels, with the intervention
only of the pericardium and some lax areolar and adipose tissue.
The dimensions of this region, which corresponds to that of abso-
lute precordial dulness, vary not only in disease, but likewise in
health; thus, whilst the lungs recede in expiration it is consider-
ably extended, and in healthy persons with well-developed
lungs it may be much contracted. In hypertrophy of the heart
unassociated with pulmonary emphysema it may be greatly en-
larged; whilst, in the last-mentioned disease, it is always much

reduced, and may be even entirely abolished. In the remainder of its extent the anterior surface of the heart is covered by the lungs. Its general outline, and the situation of its principal parts, may be conveniently indicated by the following lines and fixed points on the anterior wall of the chest. The superior boundary line of the auricles, and the roots of the great vessels connected with the base of the heart, correspond to the upper edge of the third costal cartilages. The right extremity of the base, formed by the right auricle, is indicated by a curved line, convex to the right, commencing a little to the right of the mid sternal line on a level with the upper edge of the third costal cartilages, thence passing downwards and to the right, crossing the third intercostal space and fourth costal cartilage half an inch from the sternum, and curving slightly inwards and downwards to terminate in the fifth interspace, somewhat less than half an inch to the right of the sternum. The inferior boundary of the heart may be determined by a line carried from the last-mentioned point towards the left side and slightly downwards, a quarter of an inch above the base of the xyphoid cartilage, crossing the common cartilage of the sixth and seventh ribs of the left side at its junction with the sternum, thence passing horizontally into the fifth intercostal space, to terminate in that space at an equal distance from the two adjacent ribs, beneath the junction of the fifth rib with its cartilage, two inches below the level of the nipple, and one inside the nipple line. The left boundary is mapped out by a slightly curved line, convex to the left side, commencing at the upper edge of the left third costal cartilage at the junction of its middle with its outer third, passing obliquely downwards and outwards, crossing the fourth costal cartilage half an inch inside its junction with the corresponding rib, and thence passing downwards, with a slight inclination inwards, to terminate in the fifth interspace, one inch to the right of the nipple line.

The line of the auriculo-ventricular sulcus, slightly curved, and convex to the right side, commences at the middle of the left third costal cartilage, and terminates at the junction of the sixth and seventh right common cartilage with the sternum.

The orifice of the pulmonary artery is beneath a point in the

second intercostal space, immediately to the left of the sternum, and that of the aorta, the left two-thirds of which are covered by the orifice of the pulmonary artery, is behind the lower portion of the left third chondro-sternal articulation, and the adjacent portions of the cartilage and sternum.

The centre of the left auriculo-ventricular orifice corresponds to a point on the left third costal cartilage, less than an inch from the sternum. This orifice, being remote from the surface of the chest, and covered by the great vessels, is unfavourably circumstanced for auscultation; the sounds having their origin in it are therefore most distinctly audible at the apex. In like manner, the orifice of the aorta being overlain in greater part by that of the pulmonary artery, sounds, whether normal or abnormal, originating in it, are most distinctly heard, and with the least liability to a mistake of identity, at the centre of the sternum on a level with the lower edge of the third costal cartilage, and thence obliquely upwards beneath the inner portion of the second right intercostal space and costal cartilage. The right auriculo-ventricular orifice is behind the centre of the sternum, at the level of the fourth intercostal space. The ascending portion of the arch of the aorta bulges a quarter of an inch beyond the right border of the sternum, and the vena cava superior projects half an inch outside the aorta in the first and second right intercostal spaces, and behind the second costal cartilage. [Quain and Sharpey]. The trunk of the pulmonary artery is entirely to the left of the mesian line, and its left border projects three-fourths of an inch beyond the left margin of the sternum. The right auricular appendix covers the root of the aorta to the right, and on a level with the orifice of the pulmonary artery, and its point is a little to the right of the middle line of the sternum on a level with the upper edge of the third costal cartilages. The *conus arteriosus* of the right ventricle extends from the lower extremity of the body of the sternum, obliquely upwards to the inner portion of the left second intercostal space. The apex of the left auricular appendix is one and a-quarter inch to the left of the sternum, and behind the third costal cartilage. The summit of the arch of the aorta is on a level with the middle of the manubrium sterni; and the left edge of the arteria innominata at its

origin is a little to the left of the middle line. The concavity of the aortic arch is on a level with the junction of the manubrium with the body of the sternum. [Allen Thompson.]

The axis of the heart corresponds to a line traversing the fifth intercostal space, three and a-half inches to the left of the middle line, passing obliquely upwards, backwards, and to the right, crossing at an acute angle, and in succession, the bodies of the eighth, seventh, and sixth dorsal vertebræ, and the corresponding intevertebral discs, and passing, if prolonged, through the centre of the right scapula. (*See Plate* 1, *Fig.* i. *and* ii.)

The heart is a quadrilocular organ, consisting of two auricles and two ventricles; functionally, however, it is double, being composed of a right and a left portion, which, normally, have no direct communication with one another after birth. During fœtal life a direct passage exists from the right into the left auricle, known as the foramen ovale, or of Botal, and the heart is consequently a single organ at that period of life. Each side of the heart consists of an auricle and a ventricle, freely communicating by the auriculo-ventricular opening, by which a current is permitted from auricle to ventricle, but is intercepted in the opposite direction by a valvular apparatus. The right auricle receives, through the two venæ cavæ, the venous blood of the entire body, except that from the walls of the heart, which enters it by the great coronary vein and the venulæ Thebesii. The blood thence passes into the right ventricle, whence it is conveyed to the lungs by the pulmonary artery, and after undergoing aeration, or conversion into the arterial state in these organs by the respiratory process, it is returned to the left auricle by the four pulmonary veins, and passed into the left ventricle, whence it is conducted by the aorta and its branches to all parts of the body. Having subserved the functions of nutrition and secretion in the various parts of the organism, the blood is conducted back in a venous state to the right auricle of the heart.

Such is a general and very brief sketch of the circulation, as discovered and demonstrated in the seventeenth century by the illustrious Harvey.

The right chambers of the heart are likewise anterior to those of the left side. The auricles constitute the base, and the ventricles the apex and principal portion of the heart.

Interior of the Heart.

The auricles are most conveniently laid open for examination by a crucial incision made into the external anterior surface of the right, and the external posterior surface of the left. Each is divisible into two portions, a *sinus* and an *appendix*, and lined throughout by a fine membrane, continuous with that covering the inner surfaces of the blood vessels, and named endocardium by Bouillaud, in contradistinction to the outer or investing serous membrane, the *exocardium*. This membrane belongs structurally to the serous class, being composed of two layers, viz.: on the inner or free surface an epithelial stratum, consisting of a single layer of flat, oval, nucleated, and tesselated particles, having an average long diameter of 1-1700 and a short diameter of 1-3500 part of an inch in the fœtus of six months. These are supported externally by a layer of fine connective tissue, in which the elastic element preponderates in the auricles. The auricular sinuses are smooth throughout, with the exception of a portion of the anterior wall of the right, but the appendix of each presents an uneven or reticulated surface, owing to the presence upon it of a great number of fleshy columnar projections, or pilasters, which, with a few connecting bands, are disposed in parallel lines from the apex towards the base of the appendix, and thence, in the right auricle, downwards upon the anterior wall of the sinus, as far as the auriculo-ventricular opening. In the left auricle these muscular projections are confined to the appendix, in which they are of greater magnitude than those of the right side.

The *right auricle* forms the anterior and inferior portion of the base of the heart. The appendix projects from its left and superior portion: it is irregularly conical, dentated at the margins, and curved, embracing the root of the aorta on the right side, and overlapping it somewhat by its apex.

In capacity the right auricle is generally considered somewhat to exceed the left. Its sinus is cuboid in figure, and presents on the superior wall the orifice of the descending vena cava to the right, and the narrow passage of communication with the appendix to the left side. The former is ovoid in figure, not protected by a valve, and its axis tends downwards, inwards, and somewhat forwards, in the direction of the auriculo-ventricular

opening. On the inferior wall of the sinus are seen three apertures, viz.: the auriculo-ventricular, that of the coronary sinus, and that of the ascending vena cava, placed from before backwards, and from left to right, in the order in which they have been just enumerated.

The auriculo-ventricular orifice is nearly circular in figure, and about one and a-quarter inch in diameter; it is bounded by a tendinous ring, smooth and even on the inner surface, and leads directly into the right ventricle. The opening of the coronary sinus is about three lines in diameter, and guarded by a circular or bilunate valve composed of a duplicature of the endocardium only. External to this valve is the coronary sinus, into which the great coronary, and a few minor veins open, the former guarded by a double valve.

The orifice of the inferior cava is considerably larger than that of the superior vessel of the same name; it has a direction upwards and inwards, and is guarded by the Eustachian valve anteriorly. This valve is of considerable magnitude in the foetus, serving at that period of existence to conduct the blood from the inferior cava, through the *foramen ovale*, directly into the left auricle; but at birth, when the adult circulation has been established, and its special function thereby abolished, it commences to undergo progressive atrophy, and in the adult is usually represented only by a narrow crescentic fold of membrane.*

The posterior and left wall of the auricle is formed by the septum auricularum. On this is situate a smooth prominence superiorly, intermediate between the orifices of the venæ cavæ, and named the *tubercle of Lower*. Still lower down, and immediately over the mouth of the inferior cava, a dimple or superficial depression may be seen. This is the *fossa ovalis*, the vestige of the foramen ovale already mentioned, by which, in foetal life, blood passed directly from the right to the left auricle; the closure of this passage, which occurs at birth, and signalizes the transition from the foetal to the adult state of existence, is not in all persons complete, and hence, occasionally, admixture of the venous with the arterial blood, giving rise to the condition known as *cyanosis*. The fossa ovalis is embraced in three-fourths of its circumference,

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* Senac (*opus citat*) has seen it entire as late as fifteen years after birth.

viz.: anteriorly, superiorly, and posteriorly, by a smooth, prominent, muscular crescent, the *annulus ovalis* of Vieussens. To the anterior cornu of this crescent the left extremity of the Eustachian valve is attached, an arrangement by which, in the fœtus, the passage of blood from the inferior cava through the foramen ovale is insured.

Besides the openings already mentioned, a number of minute apertures are visible, dispersed over the smooth portion of the sinus; these are the *foramina Thebesii*, the majority of which are shallow recesses in the wall of the auricle, and lined by an inflection of the endocardium ; a few, however, usually three or four in number, are the orifices of minute veins, the *venulœ Thebesii*, which open into the inferior anterior portion of the auricle. One of these is the *vein of Galen*, derived from the right border of the heart; another is that of the infundibulum of the right ventricle. M. De Lannelonque* describes four of these veins opening into the right auricle upon the interauricular septum, but subject to slight variation in number and position, One is the vein of Galen, which opens into the appendix near its right wall; another, near the superior vena cava; a third, intermediately between the orifice of the superior cava and that of the auricular appendix ; and a fourth, near the opening of the coronary sinus.

The mouths of all these vessels are funnel-shaped, and communicate freely with one another by large connecting channels beneath the lining membrane of the auricle, provided with valvular orifices. These large orifices he designates as " foramina ;" but there are generally several others of much smaller size and similar conformation dispersed over the septum, and in the vicinity of the auriculo-ventricular opening, and communicating with a fine sub-surface venous plexus, which is occasionally connected with the former. These are the formina of Vieussens and of Thebesius, and distinguished from the former by the designation of " forminula."

The *left auricle*, situated superiorly, posteriorly, and at the left extremity of the base, is bounded in front by the roots of the

aorta and pulmonary artery, the latter being overlapped on the left side by its appendix, which is somewhat longer and more curved than that of the right auricle; posteriorly, by the œsophagus, and more remotely, the thoracic aorta and other contents of the posterior mediastinum, through the medium of the pericardium; externally, by the left lung; internally and somewhat anteriorly, by the right auricle; inferiorly, by the left ventricle; and superiorly, by the left branch of the pulmonary artery, with the intervention of the pericardium. The sinus is throughout smooth on its internal surface, presenting no musculi pectinati, which are restricted to the appendix, and of much greater development than those in the right auricle; at the four angles of its posterior wall, which is somewhat convex forwards, are situate the orifices of the pulmonary veins, two on the left and two on the right side, the former usually in closer proximity than the latter, and frequently communicating with the auricle by a common orifice. These orifices are ovoid in figure, have an oblique aspect forwards and inwards, and are unprovided with valves. Several minute foramina Thebesii are likewise dispersed over the surface of the sinus, and one of large size opens on the posterior wall, between the orifices of the pulmonary veins, and conducts venous blood from the glands at the bifurcation of the trachea.

The *right ventricle* forms the inferior, anterior, and right portion of the heart, not extending, however, quite to the apex; it is crescentic in section, or rather irregularly triangular, the anterior wall being more extensive than the inferior, whilst the posterior and left wall, which is convex towards the right ventricle, is the most extensive of its boundaries, and formed entirely by the septum ventriculorum. The cavity of the right ventricle consists of two portions, the ventricle proper, constituting its greater portion, and the sinus or infundibulum, a smooth funnel-shaped passage, leading from the anterior, superior, and left extremity of the former, into the pulmonary artery. The ventricle proper is broad towards the base, and narrow towards the apex of the heart, and throughout, except at the auriculo-ventricular opening, its internal surface is reticulated by round or angular fleshy columns, the *columnæ carneæ*, which intersect at various angles, and are of three kinds, viz. : a set which are attached to

the wall of the ventricle by one surface throughout their entire length, and present the appearance of pilasters ; another, much more numerous, attached by their extremities and free in the intermediate portion; and a third, the *musculi papillares*, usually three in number, much larger than either of the former, conical in shape, connected with the ventricular wall by the larger extremity, and by the smaller end, which is mammillated and usually bifid, giving attachment to the *chordæ tendineæ*, and through these connected with the auriculo-ventricular valve. The musculi papillares arise towards the apex of the ventricle, two from the anterior wall, and the third, which is the shortest from the septum. In many hearts this third column is absent, the chordæ tendineæ in that situation arising directly from the septum. The right ventricle has two openings communicating with it, viz.: the auriculo-ventricular at thecentre of its base; and that of the pulmonary artery at the distal extremity of the infundibulum, and placed, with relation to the former, anteriorly, superiorly, and to the left side. The auriculo-ventricular orifice is provided with a valvular apparatus, which permits the flow of blood from the auricle into the ventricle, but effectually precludes reflux from the ventricle to the auricle in the normal state. This valve usually consists of three segments, and is hence named *tricuspid*; but, according to Pettigrew,* in at least thirty per cent of human hearts it is composed of two segments only, the representative of the third being rudimentary. When three segments are present, one, the largest, is situated anteriorly and somewhat to the left, between the auriculo-ventricular and arterial openings; this is the *septum of Lieutaud,* to which the function of closing the arterial orifice during ventricular diastole was for a long time attributed ; another is placed on the right side; and the third and smallest posteriorly and to the left, upon the septum ventriculorum.† The structure of these valves, and their connection with the musculi papillares and chordæ tendineæ, demand a

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* *Transactions of Royal Society of Edinburgh,* vol. xxii. part iii.

† Haller (*Elementa Physiologiæ,* tom. i., p. 291, *et sequent*) assigns to the three segments of the tricuspid valve, the relative positions, "anterior and superior," (septum of Lieutaud) "anterior and inferior," and "posterior," and, with the exception of the last, correctly, in relation to the body.

special and detailed description, owing to their intimate bearing upon the mechanism of occlusion during ventricular systole.

The *auriculo ventricular* opening is bounded by a tendinous ring, composed of white fibrous tissue, and possessing in an eminent degree the property of resistance; it is strongest anteriorly and in the vicinity of the septum, and gives attachment by its inferior and left margin to the tricuspid valve, and by its superior and right, to the fibres of the right auricle; very few of the fibres of the corresponding ventricle are attached to this ring. The valve forms an unbroken circle,* in continuity with it around the opening, and then breaks up into three segments, which occupy the relative positions already indicated. These segments are thick at the base and along the centre, and thin at the free extremity and margins, and each is composed of a reduplication of the endocardium, between the folds of which minute bands of white fibrous tissue are disposed in a definite manner.† According to Pettigrew, each branch of the bifid papillary muscles, which correspond in number with the segments of the valve, gives off the typical number of three chordæ tendineæ, each of which in turn breaks up into three, thus yielding in all nine minute tendons, which are attached to the dorsal or ventricular surface of the adjacent half of the corresponding valve; the three strongest and most superficial, at equal distances along the middle line from base to apex; the three next in point of thickness and length, to that portion of the valve intermediate between the mesial line and margin in a similar manner; and the three shortest and finest, which are likewise in situation the deepest or most remote when viewed from the ventricular aspect of the valve, are implanted into its margin, extending from the attached to the free extremity. A similar arrangement obtains for the tendons arising from the second branch of the papillary muscle,

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* In *Quain's Anatomy*, edited by Professors Sharpey, Thomson, and Cleland (seventh edition, 1867, vol. i.), the learned editors deny the existence of distinct tendinous rings at the auriculo-ventricular orifices; they assert that the so-called rings consist of bands of loose white fibrous tissue, strengthened anteriorly and towards the septum, by processes derived from the plate of fibro-cartilage, occupying the triangular interval between the aortic and the two auriculo-ventricular openings.

† Vesalius taught that the tricuspid valve consisted of a single layer of membrane.

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but which are destined for the adjacent half, not of the same, but of a neighbouring valve. At the opposite extremity of both valves, when two only are present, another papillary muscle with its two crops of tendons, is similarly disposed; but when three valvular segments exist, a third papillary muscle with its tendons, is supplied for the adjacent surfaces and margins of the second and third segments. The tendinous chords arising from a common origin, and destined for the same valve, mutually intersect at various angles, and seem to form an intricate interlacement, owing to their peculiar arrangement in layers and the varying obliquity of their courses; they all expand between the layers of the endocardium, those from opposite sides of the same segment being fused together, so as to form a dense and continuous stratum, constituting its bulk, and giving it strength and consistence. Two or three minor lobes, according to the number of the larger segments, but unprovided with papillary muscles or tendons, are occasionally interposed between the angles of the latter.

From the arrangement thus briefly described, it is manifest that each segment of the auriculo-ventricular valve is supported upon every point of its ventricular surface by two sets of tendons, proceeding from two distinct papillary muscles; that each is, therefore, influenced by two of these muscles; and, further, that each muscle acts upon two valves, and must, of necessity, in contraction, approximate their margins, and in the same proportion, tend to close the passage which they guard. Thus, these muscular columns, whose use has for a long time constituted a problem of great difficulty in physiology, perform a double function, mainly that of sustaining the auriculo-ventricular valve through the chordæ tendineæ, and of preventing its retroversion during ventricular contraction; for, being themselves offsets from, and contracting simultaneously and in an equal ratio with, the muscular walls of the ventricle, they neutralise the relaxing effect upon the valve, which must otherwise result from the gradual approximation of the apex towards the base of the heart in the progress of ventricular systole. They likewise, by their contraction, approximate the free margins of the valves, owing to the connection of *each* of

them with *two* segments, although of themselves they seem incapable of effecting complete closure, notwithstanding the high authority of Reid* and Bouillaud† to the contrary.

At the first moment of relaxation of the ventricles, the blood, now subjected to the pressure of distension in the auricle, rushes with considerable force into the ventricle, divaricating the segments of the valve, and pressing them against the ventricular walls. During the progress of ventricular diastole, however, the valves are displaced from the walls of the ventricle, and floated athwart the orifice by the accumulating blood, in such a manner as not entirely to occlude it, or prevent the further entrance of blood. The ventricle being now all but fully distended, and the pool of blood in the auricle and ventricle continuous through the partially open auriculo-ventricular orifice, the active contraction of the auricle propels a further volume of blood into the ventricle, completely distending it, and determining its immediate contraction. The pressure to which the blood is now subjected by the muscular walls of the contracting ventricle at all points of its surface, save at its base, where the walls are non-contractile, determines a movement of the mass in this latter direction, or towards the point of least resistance, with a force equivalent to the pressure exercised, which Hales‡ has estimated for the left ventricle at 7·5 feet of a vertical column of blood.

For the right ventricle, according to the calculation of Haughton, the estimate should be somewhat more than one third of the preceding.§ This pressure is borne mainly by the auriculo-ventricular valve, which is forcibly and rapidly closed, whilst the attached chordæ tendineæ, previously relaxed, are put suddenly upon the stretch,|| yielding with the valves a tension-sound, which constitutes the principal element of the first sound of the heart. The attachment of two adjacent segments, at both extremeties, to the same papillary muscle, through the chordæ tendineæ, must determine a movement of mutual approximation

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* *Cyclopædia of Anatomy and Physiology* : article " Heart."
† *Traite Clinique des Maladies du Cœur.*
‡ *Statical Essays,* London, 1769, vol. ii.
§ *Principles of Animal Mechanics,* 1873.
|| Haller *(opus citat,* tom. i. liber. iv., cor) erroneously asserts that during ventricular contraction the chordæ tendineæ are relaxed.

of their free margins under pressure from behind. I cannot by any means admit with Dr. Bell Pettigrew* that "during the systole, and towards the termination of that act, the valves are, by the contraction of the musculi papillares, dragged down by the chordæ tendineæ into the ventricular cavities to form two dependent cones;" nor with Professor Halford, that they are projected into the auricles. On the contrary, I believe that throughout ventricular systole the valves present a plane surface towards either chamber, and that, as already stated, the principal function of the papillary muscles is to neutralize, by their contraction, the relaxing effect upon the chordæ tendineæ which must otherwise follow from the approach of the apex towards the base of the heart in the progress of ventricular systole. Neither can I admit, with the first-named distinguished anatomist,† that during ventricular systole the auriculo-ventricular orifice undergoes contraction, irrespectively of the approximation of its valves; nor, that a greater degree of blood pressure is requisite for divaricating the arterial valves than for closing those of the auriculo-ventricular openings.

The orifice of the pulmonary artery is situate at the distal extremity of the infundibulum; it is circular in figure, and surrounded by a tendinous ring of greater strength and thickness than that of the auriculo-ventricular opening,‡ and guarded by a valve, the *sigmoid* or *semilunar*, consisting of three segments, one being placed in front, another posteriorly and to the right, and a third posteriorly and to the left side.§ This orifice has an average circumference in the adult male of three inches and four lines, and in the adult female of three inches three lines; whilst the appended valves, detached and expanded in close juxta-position, present a united diameter somewhat in excess of that of the orifice which they guard. The tendinous ring which encircles the arterial orifice is composed of three festoons, convex

* *On the Relations, Structure, and Function of the Valves of the Heart in Vertebrata,* *Transact. Royal Society of Edinburgh,* vol. xxiii., part iii.

† *Opus citat,* p. 797 and 799.

‡ Luton (*Nouveau Dictionnaire de Médecine et de Chirurgie Pratique,* Paris, 1868) asserts that the auriculo-ventricular zones are stronger than the arterial; the left thicker than the right; and the anterior demi-zone stronger than the posterior.

§ Haller (*opus citat*) defines the relative position of the pulmonary sigmoid valves as anterior, posterior, and superior, respectively.

towards the ventricle, and meeting at acute angles, salient towards the artery. Each of these represents about half a circle, the diameter of which is to that of the artery as 1 to 3.

To the upper or concave surface of these tendinous semi-circles the valves are attached by their convex edges, and immediately above them the artery, which is likewise attached by its middle coat to their concave surface but outer edge, expands into three recesses, corresponding in situation, depth, and figure, to the segments of the valve. These are the *sinuses of Valsalva*. When the valves are partially separated from the wall of the artery, the sinuses, bounded externally by the bulging wall of the vessel, internally by the arterial surface of the valves, and inferiorly or towards the ventricle by the curve of the semi-circular tendons, to the inner and outer edges of which, respectively, the valves and the middle coat of the artery are attached, present each the figure of a boat, and are veritable navicular fossæ.

On a line corresponding to the extremities of the tendinous curves, and the upper boundaries of the sinuses of Valsalva, the wall of the artery is uniformly thick, but that portion of it which enters into the sinuses is considerably attenuated. In the triangular interspaces between the adjacent convex edges of the tendinous festoons at the root of the artery, a dense layer of fibrous tissue, continuous with the festoon on either side, and lined by the endocardium, alone constitutes the wall of the ventricle.

The segments of the valve are thick and uniform along the attached border, but thin and double festooned on the free margin. They are composed, like the tricuspid valve, of a reduplication of the endocardium, with fibrous tissue interposed. The arrangement of this latter element deserves special attention. According to Pettigrew,* a tendinous thread of extreme tenuity passes along the free border of the valve on each side, to meet in the centre, where a nodular thickening exists, known as the *corpus arantii*. From all points of the convexity of this marginal tendon others of still greater tenuity pass off at acute angles, descending obliquely inwards, the most internal intersecting in the middle

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* *Opus citat*, p. 769 *et sequent*, and plate xxviii.

line of the valve, whilst the external descend towards its attached border, intersecting in their course those placed lower down. Between this marginal filament and the next band of magnitude beneath, the valve over an extent of several lines is thin and pliant, and composed only of the two layers of endocardium, with the offsets from the marginal tendon previously described; this is the *lunula*. Beneath this four or five strong tendinous bands arise on each side from the tendon at the attached border of the valve, and, at equal distances apart, course downwards and inwards with a gentle curve, concentric with those of the margins, and branching as they advance, to intersect those from the opposite side along the middle line; and finally, from the lower margin of the valve, at the bottom of the sinus, several small bands pass upwards and inwards on either side of the mesial line, bracing the others, and giving increased thickness and strength to the attached border of the valve.

During ventricular systole the semilunar valves are divaricated against the walls of the artery by the centrifugal pressure of the escaping column of blood; and according to Haller, the purposes of the sinuses of Valsalva is to furnish recesses in which they may be lodged at this moment, out of the course of the blood. The position of the valves, however, above the tendinous ring, and their extreme tenuity, would seem to render such a provision scarcely necessary. It seems more probable that the use of the sinuses, as urged with much emphasis by Pettigrew, has reference to the direction of closure of the valves.

At the termination of ventricular systole, the elastic recoil of the walls of the artery determines a backward movement of that portion of the column of the blood in proximity to the ventricle; by this movement the valves are closed over the opening with a suddenness and force sufficient to elicit a tension-sound, known as the second sound of the heart. The direction of the blood-pressure, by which the valves are thus closed, is so modified by the pouching of the artery at the sinuses, that if the axes of the columns borne by the three valves respectively were prolonged towards the ventricle, they would meet immediately below them in the axis of the passage. Hence the valves, pressed against one another at the moment of closure,

become mutually sustaining, and the force that would tend to effect their retroversion into the ventricle, is converted into an agency by which that accident is averted.

It has been already shown that the diameter of the conjoined valves exceeds that of the orifice, and that the lunulæ are much thinner and more flexible than the remainder of the valve; if to these two conditions be added the shortness of the marginal tendons, by which the free edge of each valve is slung up between the most elevated points of the corresponding tendinous festoon, and the convergence towards the arterial orifice of the lines of blood-pressure from above, it will be manifest that retroversion cannot occur without rupture of the valve; and further, that rupture cannot take place within the area of the lunulæ, which are disposed *dos à dos*, and out of the lines of blood-pressure. Experience proves the correctness of both these conclusions, displacement of one or more of the segments into the ventricle having never been met with in the absence of disintegration or detachment to some extent, and rupture being confined to the central and attached portions of the valves. The absence of an apparatus of muscles and tendons, destined to prevent retroversion of the semilunar valves during arterial systole, such as that previously discussed in connection with those of the auriculo-ventricular opening, may be explained in part by reference to the mechanical obstruction to the transit of the blood which would result from their presence in the narrow channel of the artery, but chiefly by the fact that such a provision is unnecessary, and would be totally inapplicable to the circumstances under which the semilunar valves are brought into action. The force by which the valves are closed in both instances is, no doubt, blood pressure; but in the one case this is the equivalent of the elastic reaction of the artery in the direction of its axis, whilst in the other it is the representative and guage of a much greater force, the active contraction of the walls of the ventricle, operating not only towards a common centre, but with a definite movement of approximation of the apex towards the valves. The form of the artery favours the application of the provision actually made for the support and protection of its valves; and experience

proves that this provision is fully adequate to its purpose, whilst the greater size and power of the ventricle would render such an arrangement not only unsuitable, but insufficient.

The left ventricle occupies the posterior, inferior, and left portion of the heart, extending to the apex, which it entirely forms; about one-third of its superficial extent enters into the anterior surface of the heart, the remaining two-thirds constituting the left border, posterior surface, and part of the inferior surface of the organ. In figure it represents a slightly flattened cone, and in section it is ovoid, the long diameter from before backwards, the septum being concave to the left. Pettigrew[*] describes the cavity of the left ventricle as circular at the base, oval half an inch from the base, irregularly triangular half an inch still lower down, more decidedly triangular two inches from the base owing to the projection of the musculi-papillaries, and finally bayonet-shaped at the apex. The walls are thickest at the junction of the basic fourth with the apicial three-fourths of its length, and here they are equal to thrice the thickness of those of the right ventricle. The endocardium is more opaque, and the columnæ carneæ are smaller, but more numerous and more closely reticulated than in the right ventricle, especially towards the apex; but on the upper portion of the anterior wall and septum they are entirely absent.

The musculi papillaries are only two in number, both arising in close proximity to the septum, one from the anterior and the other from the posterior wall of the ventricle. They are much thicker, and the chordæ tendineæ are stronger and less numerous than in the right ventricle. At the base the auriculo-ventricular and arterial orifices are situate, the former posterior and to the left, the latter anterior and to the right side, and in much closer mutual proximity than the corresponding orifices in the right ventricle. The left auriculo-ventricular orifice, smaller than the right,[†] is ovoid in figure, the long

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[*] *Philsop. Transact.*, part iii. 1864.

[†] Dr. Peacock estimates the average circumference of the auriculo-ventricular orifice as follows :

		Males.	Females.
Right		4 inches 6 lines.	4 inches 0 lines.
Left		3 „ 7 „	3 „ 10 „

diameter directed obliquely from before and from the left side backwards and to the right; it is surrounded by a strong tendon, and guarded by a valve, the *mitral*, thicker and stronger than the tricuspid, and consisting of two segments. Of these the larger, interposed between the two openings, is placed with relation to the auriculo-ventricular orifice anteriorly and to the right, whilst the other and smaller segment is posterior and to the left.*

The musculi papillaries of the left ventricle are connected through the chordæ tendineæ with the segments of the mitral valve, in a manner in no respect different from the arrangement already described in the right ventricle. The function of the mitral valve is likewise similar to that of the tricuspid; and the explanation already given as to the mechanism of closure of the latter, may be applied, *mutatis mutandis*, to that now under consideration.

The orifice of the aorta is situate at the superior, anterior, and right portion of the ventricle; it is circular in figure, somewhat smaller than that of the pulmonary artery, posteriorly and to the right of which it lies, but separated from it by the septum of the ventricles.† The sigmoid or semilunar valves, three in number, and placed with respect to the opening, one anteriorly and to the right, another anteriorly and to the left, and a third posteriorly, are much stronger, and the *corpora arantii* more prominent than those in connection with the pulmonary artery. The tendinous zone, similarly constructed, is likewise stronger. It is lodged in the anterior angular recess between the two auriculo-ventricular zones, and the triangular interval bounded by all three, is occupied in the ox by a calcareous mass, the so-called *os cordis*, which in the human heart is represented by fibro-cartilage. The sinuses of Valsalva are deeper and larger‡

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* Haller (*opus citat*) says the mitral orifice is oval horizontally, the valves being respectively "superior" and "inferior."

† Peacock's estimate of the mean circumference of the arterial orifices is as follows :

	Males.	Females.
Right (pulmonary)	3 inches 4 lines.	3 inches 3 lines.
Left (aortic)	3 ,, 0 ,,	2 ,, 10 ,,

‡ Haller (*opus citat*) states that there are no aortic sinuses in the fœtus or infant.

than those of the pulmonary artery; from the two anterior of these sinuses the coronary arteries take origin, the anterior from the left, and the posterior from the right sinus. Brücke maintains that the orifices of these vessels are within range of the valves, and covered by them when raised; whilst Haller,[*] Senac,[†] Bertin,[‡] and Morgagni, and most modern authorities, hold a contrary opinion. I agree with the latter, being convinced, from personal examination in the dissecting room and dead-house, of a great number of hearts free from disease of the aortic valves, that Brücke's opinion is incorrect.

To this subject I shall have occasion to revert when discussing the rhythm of the heart.

The muscular fibres of the heart observe an apparently complex arrangement, being disposed in successive layers, each of which affects a different course from those superficial to and those beneath it. Vesalius[§] described the fibres of the ventricles as being, some straight, some oblique, and others transverse; the deep fibres taking a course contrary to that of the superficial. He compares the arrangement to a "plait of rushes."

Lower[§] believed that from the tendinous rings surrounding the auriculo-ventricular openings, the fibres of the ventricles, disposed in two planes, a superficial and a deep, expanded over the chambers in a spiral manner; a few at the surface run straight from base to apex. The external fibres embrace the two ventricles; those which cover the right ventricle ascend from left apex to right base, and those which cover the left ventricle ascend from right apex to left base. The fibres, though somewhat spirally disposed, are not in unbroken continuity, the arrangement being that of the figure of 8, the fibres which pass in one direction form the external surface, whilst coursing in the opposite direction they line the internal surface. The most superficial fibres only pass to the apex, those more deeply seated turning inwards sooner.

Lancisi[§] held that a tendinous ring surrounded each of the four openings of the heart, and that from those of the auriculo-ventricular orifices the fibres arose and passed spirally round

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* *Opus citat,* liber. iv. cor. p. 367.   ‡ *Traite des Maladies du Cœur.*
† *Traite de la Structure du Cœur,* p. 365-6.   § Cited by Senac *(opus citat).*

the ventricles, being interlaced like wicker-work in their course. The fibres of the auricles start from the roots of the venæ cavæ and pulmonary veins, and passing over the auricles in two layers, viz., external and internal, end in being attached to the tendons of the auriculo-ventricular openings.

Senac* describes three layers of fibres in the ventricles. The most superficial arise from the veins and auricles, and pass spirally from base to apex. Several, however, dip into the substance of the heart, and seem to serve as " binding " fibres for others. The principal fibres turn in at the apex and pass upwards, forming the internal surface of the ventricles. The descending fibres incline at different angles to the axis of the ventricles, some at acute, and others at nearly right angles, the latter not reaching the apex, and all return to be attached to the tendinous rings of the auriculo-ventricular openings. Between the descending and the ascending fibres is a third layer similarly disposed, but not traceable to the base. The arrangement is the same for the two ventricles, and their fibres conjointly form the septum.

Winslow maintained that the heart was composed of two muscular sacs enclosed in a third, which was equally muscular. Haller† describes all the fibres of the ventricles as seeming to him oblique, approaching rather the horizontal than the vertical line. Arising in the situation of the right auricle, they are bent round the anterior or convex surface of the heart, to the apex, and thence back to the right auricle along the inferior or plane surface of the heart. Near the base many fibres run inwards towards the cavity to form the carneæ columnæ, and hence the walls are thinner at the apex than at the base. The external and internal strata are more oblique, the intermediate fibres more transverse. In the septum cordis the fibres of both ventricles are interlaced, but nearly parallel. The fibres of the musculi papillaries are straight, and run from apex towards venous (auriculo-ventricular) openings.

He described the superficial fibres of the left auricle as being transverse, and the deep as oblique, some ascending, some descending, and others surrounding the orifices of the pulmonary veins.

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* *Opus citat.* † *Opus citat,* liber. iv., cor.

Pettigrew* has carefully studied and minutely described the arrangement of the muscular fibres of the heart. According to him the ventricles are composed of seven superposed layers of fibres, *i. e.*, three superficial layers, a "middle layer," and three deep layers. The three superficial layers run from base to apex, and from right to left downwards, with progressively increasing obliquity, the first nearly vertical, and the third nearly horizontal, whilst the fourth or middle layer is all but horizontal. These fibres are connected at the base chiefly with the auriculo-ventricular tendinous rings, but partly also with those of the arterial orifices; a few likewise with the columnæ carneæ. They do not, however, arise from those rings, being connected with them only by areolar tissue. Several are inflected from the anterior surface, in the line of the anterior coronary groove, to form the ventricular septum, two thirds of which are formed of the inflected fibres of the left ventricle, and one-third by those of the right ventricle. In the septum the inflected fibres course obliquely upwards. The fibres of the deep layers run from apex to base, and from right to left *upwards*, likewise with progressively increasing obliquity, those of the first (fifth from the surface) being nearly horizontal, and those of the third (seventh from the surface) nearly vertical. The deep fibres are continuous with those of the superficial layers at the apex and base, so as to form a series of concentric elipses, the seventh with the first, the sixth with the second, and the fifth with the third, respectively. The layers are curtailed in vertical dimensions in progressive ratio from the first to the seventh, so that the extreme apex is composed of only a single layer, viz. the first, the anterior and posterior portion of which are so inflected and mutually involved that the anterior fibres, after curving round, enter the apex at the posterior surface, and become continuous with the posterior columæ carneæ and papillary muscle; whilst the posterior fibres, after being inflected to the front, enter the apex anteriorly, and become continuous with the anterior carneæ columnæ and papillary muscle. Thus is formed the " vortex,"

* *On the Arrangement of the Muscular Fibres in the ventricles of the Vertebrate Heart, with Physiological Remarks,* by James Bell Pettigrew, M.D., *Philosop. Trans.,* part iii., 1867.

or " whorl," at the apex of the left ventricle. The aggregate of the columnæ carneæ and musculi papillares constitute the seventh layer, which courses spirally from right to left, and from apex to base.

The right ventricle is moulded on the left. The septum ventriculorum is formed by an inflection of the anterior wall in the line of the anterior interventricular sulcus, and none of the layers cross this groove except a narrow band derived from the superficial layers near the base, whilst posteriorly all the layers cross the groove. Hence, perhaps, the deficiency of the basal portion of the septum ventriculum in the early stage of embryonic development. The inflected or septal fibres derived from the right ventricle belong exclusively to the three superficial layers; all the more deeply seated fibres being interrupted at the septum. Hence the right side of the septum is much thinner than the left.

The layers entering into the right ventricle are less thick than those of the left, a fact which explains the comparative thinness of the walls of that chamber. The walls of both ventricles are thickest at the centre, owing to the relative vertical dimensions of the successive layers previously described.

The sinuses of the auricles are composed of a superficial layer common to both auricles, and passing horizontally from one to the other, but most distinctly so on the anterior surface. A great portion of this layer is inflected into the interauricular septum posteriorly; and two deep layers, one of which consists of looped fibres connected by both their extremities with the auriculo-ventricular rings, and the other of annular fibres, situate at the venæ cavæ, the coronary sinus, and the pulmonary veins, and constituting a sort of sphincter for these vessels. Upon the venæ cavæ and the pulmonary veins these fibres extend from the auricles nearly half an inch. The walls of the appendices consist of a superficial layer of annular, and a deep layer of vertical fibres, the musculi pectinati already described.

Luton * describes the fibres of the ventricles as common and proper. The former are the " unitive" fibres of Gerdy, and are disposed on the anterior and posterior surfaces. The anterior

* *Nouveau Dictionnaire de Médecine et de Chirurgie Pratique;* Paris, 1868.

fibres pass down from right to left, and from base to apex, and are disposed in three ways, being all, however, rolled in at the apex like a whorl, leaving a minute aperture between, through which the ventricle may be entered without dividing any of its fibres. One set, the "simple looped fibres" (Bourgery), pass from the anterior to the posterior wall, and return to the base along the internal surface of the posterior wall, to be inserted into the posterior segments of the aortic and mitral zones. Another set, the "figure of 8" fibres (Bourgery), pass from behind forwards at the apex, course along the inner surface of the anterior wall to the base, where they are likewise attached to the anterior segments of the aortic and mitral zones. The third set passes in to form the columnæ carneæ, and are inserted through the chordæ tendineæ into the segments of the mitral valve.

The posterior "unitive" fibres pass from the posterior segments of the auriculo-ventricular zones, obliquely downwards and to the right edge of the heart, where they enter the right ventricle in succession, along the entire right border, the longest being at the apex, and are thence reflected upwards on the inner surface of the anterior wall, forming " simple loops," to be inserted into the right auriculo-ventricular zone, and that of the pulmonary artery, and also forming columnæ carneæ to be inserted through the chordæ tendineæ into the tricuspid valve.

The proper fibres of the ventricles are disposed in loops attached to the anterial and auriculo-ventricular zones, and form cylinders, open at the apex (but here narrow), and at the auriculo-ventricular orifice, and interposed between the descending and ascending layers of the "unitive" fibres. The septum is formed of fibres derived from both ventricles, but chiefly the left.

The auricular walls are likewise composed of two layers of muscular fibres, viz., a common and a proper.

The muscle of the heart, according to Kölliker,* differs from voluntary muscle in the following particulars, viz., the fibres are only one third the size of those of voluntary muscle ; the longitudinal striæ are occasionally better marked than the transverse ; they are more easily broken up into filaments and frag-

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* *Traité d'Histologie Humaine.*

ments. The sarcolemma is difficult of demonstration, save by reagents. The primitive fasciculi are often infiltrated with fatty granulations disposed in lines, a condition which exists in an exaggerated form in fatty degeneration. The fibres are not, as in voluntary muscle, disposed in fasciculi of different orders, but are closely packed, with very little connective tissue, and are connected with one another by branched fibres, which justifies the conclusion that they contract simultaneously. I have found the average diameter of the fibres to be 1-1300 of an inch; but the linear arrangement of highly refractile granules within the fibres I have not met with, save in the early stage of fatty degeneration. This "granular" condition of the fibres is regarded by Ormerod * as precursory to fatty transformation.

The arteries of the heart, named *coronary* from their peculiar disposition in the form of circlets or chaplets, are usually two in number, the *right* or *posterior*, and the *left* or *anterior*. They arise from the aortic sinuses of Valsalva, immediately above the level of the free edges of the valves when raised against the walls of the vessel. The former from the anterior and right, and the latter from the anterior and left sinus.

Occasionally both vessels arise by a common trunk, which subdivides into two branches, representing the usual arteries. Still more rarely there are three, or even four parent trunks, the supernumerary vessels arising as duplicates from the anterior sinuses, but smaller in size than the representatives of the usual vessels.

The right coronary artery descends between the root of the aorta and the right auricular appendix; it then enters the right auriculo-ventricular groove, passes horizontally to the right, and divides into two branches. Of these the smaller descends obliquely to the left, along the right border of the heart to the right apex, whilst the larger or horizontal branch continues in the auriculo-ventricular groove, to the posterior interventricular sulcus, where it undergoes further division into two branches, viz., a vertical or larger, which descends in that groove to the apex, and a horizontal, which terminates by anastomosing with the horizontal branch of the left coronary artery. In its course the

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* *St. Bartholomew's Hospital Report,* vol. iv., 1865.

trunk of the right coronary artery supplies small branches to the right auricular appendix, pulmonary artery, right auricle and ventricle, whilst its branches are expended in supplying the right auricle and ventricle, and the posterior part of the septum ventriculorum.

The left coronary artery descends obliquely to the left, between the pulmonary artery and appendix of the left auricle, to the anterior interventricular groove, where it undergoes division into two branches, the larger of which descends in that groove to the right of the apex, where it inosculates with the vertical branch of the right coronary,* whilst the smaller takes a horizontal course in the left auriculo-ventricular sulcus, to anastomose posteriorly with the corresponding branch of the right artery. This branch supplies by its trunk some branches to the aorta, pulmonary artery, and left auricular appendix, and by its two branches of division, the left auricle and ventricle and the anterior portion of the septum.

The coronary veins are divisible into the *great cardiac* or *coronary vein*, and the small coronary veins. The great coronary vein commences at the apex of the heart, whence it ascends by the anterior interventricular furrow, receiving in its course superficial and deep branches from both ventricles, and from the septum. At the base of the ventricles it becomes horizontal, passing in the left auriculo-ventricular groove to the posterior surface of the heart, and opening into the right auricle between the orifice of the inferior vena cava and that leading into the right ventricle. This vessel, in the terminal portion of its course, is remarkable for its large size, and named the *coronary sinus*; it receives tributary branches from the left auricle and ventricle; amongst the latter is a large branch which ascends along the left border of the heart to join the principal trunk at a right angle, and a still larger vein, the posterior interventricular, which ascends from the apex by the posterior interventricular groove, and opens into the coronary sinus at its termination.

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* Hyrtl (*Nat. Histor. Review*, 1861, p. 321) as the result of a series of experiments with injection, declares that the communication between the right and left coronary arteries, if it exist at all, is not such as to admit of the passage of ordinary injection material from one to the other.

The coronary veins are unprovided with valves, save at their openings into the sinus. The communication of the sinus with the right auricle is guarded by the valve of *Thebesius* already described. Cruveilhier[*] denies the alleged competency of this valve to prevent reflux, because, in his experience, injection thrown into the auricle by the superior vena cava has always found entrance into the coronary sinus. The contraction of the auricle, however, at the only period when the contained blood is subjected to pressure equivalent to that of an injecting syringe, is probably sufficient to insure the closure of the orifice and prevent reflux. The small coronary veins are those which have been previously described as conveying blood, chiefly from the right side of the heart, and opening by minute orifices into the right auricle.[†]

The *nerves of the heart* are derived immediately from the cardiac plexuses, two in number, viz., the superficial and the deep. These plexuses are formed by the union of the *cardiac* branches derived from the three cervical ganglia of the sympathetic, and those from the pneumogastric and the recurrent laryngeal nerves.

The cardiac nerves from the sympathetic are three in number, the superior, middle, and inferior. The *superior* or *superficial* cardiac nerve arises by two branches from the superior cervical ganglion, and by an equal number from the sympathetic cord below it; it descends the neck, resting on the longus colli muscle behind the carotid sheath, and inside the middle cervical ganglion; at this point it gives off a large branch which undergoes division into three. One of these crosses over the inferior thyroid artery, and descends to the thyroid body, whilst the two remaining branches embrace the common carotid artery, and descend on its anterior surface to join the first cardiac branch of the pneumogastric. The nerve then passes across the inferior thyroid artery and recurrent laryngeal nerve, and on the right side enters the chest either before or behind the subclavian artery, and is conducted by the arteria innominata to the arch of the aorta, behind which it passes to join the posterior cardiac

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[*] *Descriptive Anatomy,* vol. ii., p. 766.

[†] Senac denies that any of these veins open into the left side of the heart.

plexus, giving off a few branches which twine around to the anterior surface of that vessel.

The nerve of the left side enters the chest, resting on the left common carotid artery, and crosses the arch of the aorta anteriorly or posteriorly (usually the former) to enter the anterior cardiac plexus. These nerves, in their course, communicate with the external laryngeal, the pneumogastric, the middle cervical ganglion, and the recurrent laryngeal.

The *middle* cardiac nerve, also named *deep* and *great* cardiac nerve, arises from the lower and inner angle of the middle cervical ganglion ; it then descends behind the carotid sheath, and enters the chest on the right side by passing either in front of or behind the subclavian artery*; and on the left, between the carotid and subclavian arteries. In its course down the neck it communicates with the superficial cardiac, and the recurrent laryngeal nerve, and after entering the chest follows the course of the trachea, by which it is conducted to the deep cardiac plexus.† In the thorax, the deep cardiac communicates with the recurrent laryngeal and the superficial cardiac nerves, the connection with the latter being established, on the right side, on the posterior and external surface of the innominate artery.

The *inferior* cardiac nerve arises from the lower cervical, frequently also in part from the first thoracic ganglion; on the right side it descends behind the subclavian artery; and on the left, in company or in union with the middle cardiac nerve, to enter the deep cardiac plexus.

On both sides this nerve communicates with the middle cardiac and the recurrent laryngeal; and on the right, this is observed to take place behind the subclavian artery.

The cardiac branches of the pneumogastric are derived in part directly from that nerve, and in part from the recurrent laryngeal. The former are, in the neck, *superior* and *inferior*. The *superior* cardiac branch or branches of the pneumogastric arise from that nerve about the level of the thyroid cartilage, and after a short course downwards, are lost by uniting with the

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* Scarpa (*opus citat.*) describes it as invariably passing in front of the subclavian artery.

† According to Scarpa (*opus citat.*) it is lost in the cardiac ganglion of Wrisberg.

sympathetic cardiac nerves.* The *inferior* cardiac is a nerve of considerable size, arising from the pneumogastric at the lower part of the neck; on the right side it enters the chest on the outer side of the innominate artery, and joins one of the cardiac branches of the sympathetic, which it accompanies into the deep cardiac plexus. The inferior cardiac nerve of the left side passes in front of the aorta to enter the superficial cardiac plexus. Finally, within the chest, the right pneumogastric gives off, whilst lying on the side of the trachea, a few small branches which descend to the deep cardiac plexus; but on the left side these branches come from the recurrent laryngeal; which nerve also gives off on the right side several cardiac filaments from the convexity of its curve.

The superficial cardiac plexus lies within the curve of the arch of the aorta, and in front of the right branch of the pulmonary artery. It is formed from the left superior or superficial cardiac nerve, which ends in it wholly or in greater part; and the inferior cardiac branch of the left pneumogastric, and occasionally also that of the right side. At the point of union of the two nerves which contribute to form this plexus, a small gangliform enlargement, the *cardiac ganglion of Wrisberg*, is occasionally found. This plexus ends in the anterior coronary plexus; but from it pass numerous lateral branches in front of the left pulmonary artery, to the left anterior pulmonary plexus.

The *deep cardiac plexus* lies between the transverse portion of the arch of the aorta, and the bifurcation of the trachea, above the point of division of the pulmonary artery. In it terminate the cardiac branches of the cervical sympathetic, except the left superior; and those of the pneumogastrics, except the left inferior; besides those derived from the recurrent laryngeal nerves of both sides. From the right side of this plexus a great number of branches descend in front of the right pulmonary artery, to join the branches from the superficial plexus in the

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* Scarpa (*opus citat.*) describes two superficial cardiac nerves as arising from the pneumogastric on each side; the superior a short distance above the subclavian artery on the right side, and passing down in front of that vessel at its origin; the inferior likewise above the subclavian artery on the right, and entering the chest in front of it.

formation of the anterior coronary plexus; from it, likewise, some branches pass behind the pulmonary artery, to supply the right auricle, and a few to join the posterior coronary plexus; and from its left side a few filaments pass along the *ductus arteriosus* into the superficial plexus, whilst the greater number of branches enter the posterior coronary plexus. From both sides of the deep plexus several branches are given off to the anterior pulmonary plexuses.

The *coronary plexuses* correspond in number and position with the coronary arteries, the course of which they follow on the surface of the heart.* The *anterior* coronary plexus is derived primarily from the superficial cardiac; it is placed between the pulmonary artery and aorta, below which point it is reinforced from the deep cardiac, and sends off numerous branches which accompany the anterior coronary artery in its course.

The *posterior* coronary plexus is formed chiefly from the left, but receives a few branches from the right extremity of the deep cardiac plexus. It accompanies the posterior coronary artery to the posterior surface of the heart.

Nerve filaments fiom the coronary plexuses accompany the branches of the coronary arteries, not only on the surface, but likewise into the substance of the heart. They are distributed in the muscular tissue of the organ, and form a fine net-work beneath its lining membrane, easily seen in the heart of the lamb.

These nerve-fibres freely intercommunicate in the substance of the heart, and at their points of junction minute gangliform enlargements, first described by Remak, † may be observed. These ganglia are dispersed everywhere through the muscular tissue of the heart, but may be found in greatest number in the interventricular and the auriculo-ventricular septa. Three of these demand special notice, viz., the ganglion of Remak *par excellence*, near the opening of the inferior vena cava; that of Bidder near the mitral orifice; and the ganglion of Ludwig in the wall of the right auricle.

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* Scarpa (*Tabulæ Neurologicæ*, Ticini, 1794, *passim*) in his description of the coronary arteries and plexuses, transposes both. Thus, he describes the right coronary artery and plexus as *anterior*, and the left as *posterior*.

† Müller's *Archiv.* 1844.

The sub-pericardiac ganglia (so called) of Lee are generally regarded as only thickenings of the neurilemma. To the ganglia in the septa between the auricles and ventricles, in the immediate vicinity of the auriculo-ventricular openings, the experiments of Paget have directed special attention, as intimately associated with the rhythmic action of the heart.

The *lymphatics* of the heart are deep and superficial. The former permeate the substance of the heart and terminate in the latter, which follow the course of the blood vessels from the apex towards the base, and there form by their union two large trunks, one for the right, and another for the left side. The right lymphatic trunk ascends, resting upon the aorta, and crosses the transverse portion of the arch between the innominate and the left carotid arteries, to the trachea, and thence to the right lymphatic duct, which it enters.

The left trunk courses along the trunk of the pulmonary artery to its bifurcation. It then passes behind the arch of the aorta to the trachea, which conducts it into the neck, where it opens into the thoracic duct near its termination. These two trunks in their course along the aorta and pulmonary artery, form, each, by their frequent division and reunion, a chain rather than a single vessel.

The *dimensions, weight, capacity,* and *thickness* of the heart, and its various parts, are subject to such considerable variety, even within the limits of health, that it is difficult, and in the opinion of many authors impossible, to fix a standard of comparison. The following estimates are given as the *means* of a great number of experiments by the subjoined authors.

Measures of size are given in inches and decimals of an inch ; measures of capacity in ounces and decimals of an ounce.

Bizot's measurements, with the exception of the depth of the sigmoid valves, are given as rendered into British measure in Dr. Stokes's work on *Diseases of the Heart and Aorta.*

	Bouillaud.	Bizot.	Gendrin.	Cruveilhier.	Lobstein.	Marshall.	Quain and Sharpey.	Reid.	Peacock.	Hayden.
Length, …	3·82 in.	3·82 in. male, 3·64 in. female	3·98 in.	—	5·5 in.	5 in.	5 in	—	—	4·25 in.
Weight, …	8·37 oz.	—	8·5 oz.	7 to 8 oz.	9 to 10 oz.	male 10 to 12 oz, female 8 to 10 oz.	—	male 11 oz, female 9 oz	male 9·75 oz, female 9 oz.	9 to 10 oz.
Thickness:										
Right ventricle …	0·21 in.	At base. Male 0·16in. Female 0·15in.	0·20 to 0·31 in.	—	0·18 in.	—	—	—	—	0·25 in.
Left ventricle, …	0·58 in.	0·43in. 0·36in.	0·39 to 0·47 in.	—	0·58 in.	—	—	—	—	0·50 in.
Right auricle, …	0·08 in.	—	—	—	0·04 in.	—	—	—	—	0·08 in.
Left auricle, …	0·12 in.	—	—	—	0·08 in. at base	—	—	—	—	0·12 in.
Capacity:										
Right ventricle,	a pullet's egg	Length. Male 3·38in. Female 2·97in. Breadth 7·40in. 6·80in.	—	right somewhat less than left	—	—	2 oz.	—	—	8 oz.
Left ventricle	a little less	Length. Male 2·61in. Female 2·83in. Breadth 4·72in. 4·10in.	—	right a little larger than left	—	—	2 oz.	—	—	8 oz.
Auricles,	equal to ventricles, right ⅓ larger than left	—	—		—	—	—	—	—	with ends of veins superadded, equal to ventricles.
Transverse diameter	3·62in. below base	—•	—	—	—	—	—	—	—	3·5 in.
Circum. at base of ventricles,	8·75 in.	—	—	—	8 in. at base	8·5 in.	8·5 in.	—	—	—
Circum. of orifices:										
Right auriculo-ventricular,	3·88 in.	male 4·81 in. female 4·18 in.	—	—	—	—	—	—	male 4·50 in. female 4·00 in.	5·49 in.
Left auriculo-ventricular,	3·54 in.	male 4·29 in. female 3·61 in.	—	—	—	—	—	—	male 3·58 in. female 3·88 in.	8·92 in.
Right arterial, …	2·66 in.	male 2·79 in. female 2·60 in.	—	—	—	—	—	—	male 3·33 in. female 3·25 in.	2·74 in.
Left arterial, …	2·42 in.	male 2·74 in. female 2·49 in.	—	—	—	—	—	—	male 3·17 in. female 2·83 in.	2·35 in.
Depth of sigmoid valve	—	0·55 in. At middle	—	—·	—	—	—	—	—	0·46 in. pul. a. 0·41 in. aortic
Thickness of inter-ventricular sept	0·91 in.	male 0·43 in. female 0·39 in.	—	—	—	—	—	—	—	0·41 in.

Doctor Herbert Davies,* in a communication to the Royal Society, lays down a law of fixed relative proportion between the areas of the orifices on the two sides of the heart, which may be briefly stated as follows, viz.: As an indispensable condition to the maintenance of the circulation, and the avoidance of stasis, equal volumes of blood must pass through the corresponding orifices on the two sides of the heart in the same time, or during the systole of the auricles and of the ventricles respectively; but as the systemic is more extensive, and liable to greater and more numerous obstacles than the pulmonic circulation, it must be effected with greater velocity and force than the latter, in order to be accomplished within the same time, and with equal certainty, and the left ventricle must exceed the right in thickness in an equal proportion; but in order that the same volumes of blood shall pass through corresponding orifices within the same period of time, but with different velocity and force, the areas of the orifices must differ in a degree correlative with that of the velocity and force of the circulation through them, but *inversely*; and furthermore, the relative proportion of the area of the corresponding orifices on the two sides of the heart is the same; thus, the area of the tricuspid : area of the mitral orifice : : area of pulmonic : area of aortic orifice. The velocity of the streams through the respective orifices is inversely as the areas of those orifices. Through the tricuspid orifice the velocity of the current is 5 inches per second, through the mitral 7 inches; pulmonic, 7·3 inches, and aortic 23·1 inches in the second.

This is a most important communication, as placing upon a proper and scientific basis the relative size of the corresponding orifices, and thickness of the walls of the corresponding chambers on the two sides of the heart.

The proportion which the weight of the heart bears to that of the body has been variously estimated; by Tiedemann it is given as 1 : 160; by Clendinning as 1 : 158 (male), 1 : 149 (female); and by Reid as 1 : 169 (male), 1 : 176 (female).

Bouillaud† states that the walls of the heart increase in

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* *Transactions of Royal Society,* 1870, and Abstract in *Med. Press and Circular,* 27th April, 1870.

† *Traité Clinique des Maladies du Cœur,* tome premier, 1835.

thickness in a progressive ratio from 16 to 40 years, being thicker in persons of strong build and tall stature than in those of the opposite conformation.

According to Reid[*] the heart increases in weight during the entire period between 16 and 70 years, being in the male 8oz. 10dr., and in the female 6 oz. 13dr. at the first-mentioned age, and 12oz. 6dr. and 9oz. 5dr. respectively, at the age of 70 years. Peacock's[†] estimates for these ages respectively, in the two sexes, are, male 8oz. 2⅔dr., female 8oz. 1⅔dr.; and male 10oz. 13⅓dr., female 7oz.

Bouillaud estimates the weight of an *atrophied* heart as in the mean 175 grammes below the standard, and an *hypertrophied* heart as having a mean excess in weight of 211 to 298 grammes. The most hypertrophied heart, he adds, is more than five times the weight of the most atrophied, and nearly thrice that of the normal heart. The heaviest heart which he has met with weighed 21½oz. Lobstein has seen one which weighed 32oz. after removal of clots. I have in my possession two hearts, to be referred to in the sequel, each of which weighs over 32oz.; one of these was removed from the body of a boy under eighteen years of age. Dr. Stokes[‡] has exhibited to the Pathological Society a heart, with adherent pericardium, which weighed 4lbs. 2oz.

The following graphic account of the *development* of the heart and great vessels is taken from *Quain's Anatomy* :[§]

"The heart first appears as an elongated sac or dilated tube lying at the forepart of the embryo, having two veins connected with it behind, and a large arterial trunk proceeding from it in front. This tube exhibits rhythmic contractions of its walls from a very early period. Its form is at first symmetrical, but soon it becomes curved or bent upon itself like a horse-shoe, and projects on the ventral aspect of the body towards the right side.

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* *Lond. and Edinb. Monthly Jour. of Med. Science,* April, 1843.

† *Ib.,* 1846, and subsequently (1854) printed in separate form.

‡ *Pathol. Society* (Dublin) *Reports,* 1868-9.

§ *Elements of Anatomy,* edited by Drs. Sharpey, Allen Thompson, and Cleland, 1867, vol. i. p. 323.

" As this bending increases, the venous end approaches the arterial, and at the same time the tube, which progressively increases in size and in the thickness of its walls, becomes divided by two slight constrictions into three compartments, opening successively into each other. The first, next to the veins, is the *auricular* portion, the middle one is the *ventricular*, and the last, which is the primitive arterial trunk, is named the *bulbus arteriosus*.

" The auricular portion becomes placed behind the ventricular compartment, and relatively to that cavity, considerably enlarged. Moreover, two little pouches appear upon it, one at each side, which form the future auricular appendages. The walls of the ventricular portion are already thicker than the rest.

" The next series of changes consists in the gradual subdivision of the single auricle, ventricle, and arterial bulb, each into two compartments, to form the right and left auricles, the right and left ventricles, and the pulmonary artery and aorta ; and these changes are accompanied by an alteration in the position of the parts with relation to the body, the ventricular portion now lying transversely, so as to bring that portion which is afterwards to form the apex, towards the left side.

" This subdivision commences first in the single *ventricular* portion of the heart. A small notch appears externally to the right of the apex, which goes on increasing in depth for some weeks, and then is again gradually obliterated. In the meantime, about the fourth or fifth week, a septum begins to rise up internally from the right side of the heart, at a little distance from the apex, and from the anterior wall of the cavity, and proceeds in the direction of the base, towards the arterial bulb, and about the eight week is complete. Traces of the subdivision of the auricular portion commence early in the form of a slight constriction on the outer surface, which marks off the future auricles, the left being at first the smaller of the two but the auricular septum is not begun until after that of the ventricles is completed. About the ninth week it appears, growing from above and behind downwards and forwards, and at length comes to meet and coalesce below with the rising edge

of the interventricular septum. The interauricular septum, however, remains incomplete during intra-uterine life, and leaves an opening in the middle which forms the *foramen ovale*. The farther steps in the separation of the auricles are connected with the changes which take place at the entrances of the great veins. There are now three large vessels terminating in the auricular extremity of the heart. Of these, two correspond with the superior and the inferior vena cava, and the third is the great coronary vein. At first, after the interauricular septum is partly formed above, the inferior cava opens directly into the left auricle, which is the smaller of the two, but about the twelfth week a septum, *the valve of the foramen ovale*, which afterwards forms the floor of the fossa ovalis, rises up on the left side of the entrance of the vein, which thus comes to open into the right auricle; whilst at the same time the separation of the two auricles is also rendered more complete by the gradual advance of the valve over the foramen ovale, leaving, however, the passage open until after birth.

" Another valvular fold is developed at an early period on the right and anterior border of the orifice of the inferior cava, between it and the auriculo-ventricular orifice; this is the Eustachian valve. It appears to continue the opening of the inferior cava towards the upper margin of the foramen ovale, and directs the blood of the vein through that passage.

" The left auricle has at first no connection with the pulmonary veins. The manner in which the connection is afterwards established has not yet been ascertained.

" Originally the heart is composed of a mass of nucleated cells, similar in character to those which primarily constitute the other organs of the body. Muscular tissue is subsequently formed from these cells; but the rhythmic contractions commence and proceed for some time, whilst the heart is yet composed of cells, and before the muscular fibres have been developed.

" *The Great Vessels*: At first the bulbus arteriosus is divided into two arches, which pass upwards and outwards, one on each side, then turn downwards, and form a right and left root of the aorta, which are at first separate, but afterwards unite behind the

heart and in front of the vertebral column, to form the single stem of the descending aorta. The distance soon elongates between those arches and the arterial bulb, and four other pairs of arches appear in series from above downwards, passing outwards from the vessel which ascends to the first arch, and opening into that which descends from it. Thus there are on each side *five arches*, an *internal* or *anterior* trunk uniting the origins of the arches, and an *external* or *posterior* trunk uniting their terminations, and continued into one of the *roots of the aorta*. These vascular arches are placed each in one of the branchial processes of the dorsal plates, but it is to be noted that the whole five arches do not co-exist; for the highest disappear before the las t are developed. This arrangement of blood-vessels, together with the originally single condition of the heart, corresponds to a certain extent with the permanent condition of the heart and branchial arteries in fishes, with difference as regards the vascular arches in the human fœtus, and that of mammals, birds, and scaly reptiles, that they never present any further branchial subdivision.

"As the interventricular septum is approaching the base of the heart, that is, about the seventh or eighth week, the arterial bulb becomes also divided by an internal partition, meeting from opposite sides, into two vessels, which are slightly twisted on each other, and are so adjusted as to become connected, the anterior with the right, and the posterior with the left ventricle; these vessels afterwards constitute the commencement of the pulmonary artery and of the aorta. A furrow subsequently, beginning on the outside, completes the separation into two vessels.

"Whilst the arterial bulb is thus converted into the commencement of the pulmonary artery and aorta, the five vascular arches arising from it undergo a metamorphosis, by which the permanent aorta, with the brachio-cephalic vessels and the pulmonary arteries, are formed. The general results of this change have been observed by several embryologists, but it has not yet been made out with certainty in all its details.

"It is generally admitted, however, that the fourth arch on the left side (counting from above), which receives blood from the

aortic division of the bulb, is persistent, and continuing to en-
large, eventually becomes the arch of the aorta. The fourth
arch on the right side, as well as the first, second, and third
arches on both sides, are obliterated to a greater or less extent,
while certain portions of them, remaining pervious and connected
with the aortic arch, appear to form the commencement of the
great vessels rising from it.

" Both the arches of the fifth pair were held by Baer to be
connected with the pulmonary division of the bulb, and to send
ramifications into the lungs, so as to form the right and left
branches of the pulmonary artery respectively; the further or
distal portion of the right arch being obliterated, while the cor-
responding part of the left side continued open, as the *ductus ar-
teriosus*, until birth. According to this view, the third arch on
each side is persistent as the subclavian artery, and the external
trunk above this remains as the vertebral artery, and the in-
ternal as the carotid; while the internal trunk between the third
and fourth arches of the right side becomes the innominate ar-
tery. In so far as it applies to birds and some reptiles this view
may be correct. But a different view of the metamorphosis, as
it occurs in mammalia and man, has more recently been pre-
sented by Rathke, which has been adopted by Kölliker and
others, and probably is more consistent with truth. According
to Rathke, in man and mammalia one arch only, viz., the left
fifth, is concerned in the formation of the pulmonary arteries;
and the fifth arch of the right side is entirely obliterated. From
the fifth left arch a branch is given off, which, together with
the proximal part of the arch, forms the pulmonary artery, and
which divides into the primary branches for the right and left
lung, the distal part of the arch being converted, as according to
Baer's theory, into the ductus arteriosus. The fourth arch of the
right side, according to Rathke, forms the commencement of the
right subclavian artery ; a branch is given off opposite the ex-
ternal extremity of the fourth arch on both sides, which forms
on the right side the remainder of the right subclavian, and on
the left the whole of that artery ; the vertebral arteries are de-
rived from the subclavians external to the system of arterial
arches; the internal trunks in their extent between the third

and fourth arches remain as the common carotids, and in the remainder of their extent form the external carotids, while the third arches and the external trunks above them are converted into the internal carotid arteries."

The *heart of the fœtus* occupies a vertical position in the body till about the fourth month, when it commences to undergo a partial revolution on its antero-posterior axis, by which the apex is carried to the left of the mesial line, and the long axis of the organ becomes oblique in relation to it. Again I quote from *Quain's Anatomy.** "For a long period the auricular portion is larger than the ventricular, and the right auricle is more capacious than the left; but towards birth these peculiarities disappear, and the ventricular portion becomes the larger part of the heart. As to the ventricles themselves, the right is at first the smaller; afterwards it becomes the larger of the two, and at birth their size is about equal. In the right ventricle the infundibulum is at first less marked than afterwards.

" For a time the walls of the ventricles are comparatively speaking very thick, and the thickness of both is nearly the same."

The comparatively greater thickness of the walls of the right ventricle in the fœtal heart, than in that of the adult, is due to the circumstance that in fœtal life the right ventricle has imposed upon it in great part, the function of propelling the blood through the vessels of the lower half of the body, and likewise through the extensive and tortuous circuit of the placental system; whereas, during extra-uterine existence, the pulmonary circulation alone is maintained by this chamber. The salient distinctive features of the fœtal heart are, the direct communication between the right and left auricles through the foramen ovale, the patency of the ductus arteriosus, and the large size of the Eustachian valve.

The *foramen ovale* occupies the lower and posterior part of the interauricular septum; it attains its greatest magnitude about the sixth month of fœtal life, is bounded by a smooth and rounded muscular rim, from the lower and posterior portion of which a valvular fold, the *operculum*, of a crescentic figure, and consist-

* Vol. i. p. 327.

ing of a duplicature of the endocardium with some intervening fine elastic tissue, ascends. The operculum adheres to the lateral boundaries of the foramen, but superiorly it is unattached, and here it presents a concave margin, which, although overlapping the superior boundary of the foramen, being placed on the left side of the septum, admits the passage of a current from right to left, but resists one in the opposite direction. Senac[*] correctly holds that the foramen ovale is kept open in the fœtus by relatively greater pressure on the right side of the operculum; after air has entered the lungs, however, and the pulmonary circulation has been established, the pressure borne by the two surfaces is equal, and hence immobility, and subsequent adhesion of the valve.

Usually within a period of ten to twelve days after birth complete adhesion takes place, and direct communication between the two auricles is thereby entirely and permanently interrupted; but occasionally, although closure of the passage by adhesion has not been effected, by default of nutritive activity in the tissues, or from some other and unknown cause, interauricular communication is not set up, owing, probably, to an equality of pressure existing upon the opposite surfaces of the operculum. Should this equality, however, become deranged from any cause, then intermixture of the venous with the arterial blood may take place, for the first time, it may be, at an advanced period of life.

There is another condition of the valve which may give rise to the passage of blood in the opposite direction, namely, from the left into the right auricle, of which examples will be adduced in the progress of this work; this condition is one, not of defect of adhesion but of development; the upward growth of the valve has fallen short of the superior margin of the opening, and hence patency of the latter, and transit of blood in the direction of least pressure, or from left to right side.

The existence of a third branch of the pulmonary artery, the *ductus arteriosus*, constitutes another distinctive peculiarity of the fœtal circulatory system. This branch arises from the root and distal wall of the left division of the pulmonary artery, and in point of size and direction represents the continuation of the

[*] *Opus citat.*, p. 412.

parent trunk of that vessel. It is about half an inch in length, passes upwards and to the left, and joins the aorta on its inner surface, at the termination of the arch, that is, a short distance below the point of origin of the left subclavian artery; here the left recurrent laryngeal nerve hooks round it inferiorly, in its ascent to the larynx.

During fœtal life, the great bulk of the blood circulating through the trunk of the pulmonary artery passes by the ductus arteriosus into the descending aorta, two minute streamlets only passing by the lateral branches of that vessel into the lungs, whence it is returned to the left auricle by the pulmonary veins. At birth, circulation through the ductus arteriosus is arrested; the vessel begins to shrink, and about the fourth day is completely closed; thenceforward it is represented by an impervious ligament, or fibrous band.

Doctor Alvarenga of Lisbon,* in a pamphlet of great value, gives the results of his examination of the hearts of infants and children between the ages of one day and twelve years, at the Hospital of St. Joseph, and the Foundling Hospital in that city, with a view to determining, 1st, the time of closing of the foramen ovale; 2nd, that of the ductus arteriosus; 3rd, the relative times of closing of both these structures in the same individual. The total number of hearts examined was 213, and the conclusions at which he has arrived may be summed up as follows, viz.:

1. That the foramen ovale never closes before two and a-half months, and only very rarely indeed before the age of six months, and that it frequently is found open up to the twelfth year.

2. That the arterial canal is never obliterated before the thirtieth day, and very rarely before the sixtieth, and that it is usually closed before the end of the fifth year.

3. That the arterial canal is usually obliterated before the foramen ovale is closed.

The reviewer very justly observes, in relation to Dr. Alvarenga's tables: "Much remains to be cleared up concerning the condition of the foramen ovale between that period (six months)

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* *Brit. and Foreign Med.-Chirurg. Review*, No. lxxxix, January, 1870.

and the twelfth year, before we can have any accurate knowledge of the time of closing of this structure."

Senac erroneously concludes that the closure of the ductus arteriosus is due to "extension," consequent upon the elongation of the aorta by the larger volume of blood circulating through it after the establishment of respiration.

The Eustachian valve, at a very early period of intra-uterine existence, was so amply developed as to cover completely the interval between the orifice of the inferior vena cava and the foramen ovale, and form, with the posterior wall of the auricle, a continuation of the cava quite into the foramen, so as effectually to preclude intermixture of the current entering the auricle by the inferior cava, with those from the superior cava and coronary vein. There seems reason to conclude, however, that for a considerable period before birth the valve has been incompetent to effect this object, and that its free margin has become gradually more and more distanced from the annulus ovalis, partly in consequence of the progressive expansion of the auricle, and partly because of the gradual atrophy from disuse, of the valve itself. The closure of the foramen ovale, which takes place at birth, entirely and permanently suspends this function of the Eustachian valve, which seems thenceforward to serve only as a partial "break" to reflux upon the liver in the erect posture of the body.

In the body of the fœtus, the internal iliac arteries of the adult, under the name of *hypogastric and umbilical arteries,* and in point of magnitude and direction representing the continuation of the common iliacs, pass upwards in the posterior and the lateral false ligaments of the urinary bladder, to its superior fundus, and thence in the superior or suspensory ligament or fold of peritoneum, to the umbilicus, where they meet and twine around the umbilical vein, and by it are conducted to the placenta. These vessels convey the largest portion of the blood of the common iliac arteries to the placenta, where it undergoes oxidation, and whence it is returned by the umbilical vein to the liver. The division and deligation of the *funis* after birth involves closure of all three vessels, the consequence of which, as regards the hopogastric arteries, is that these vessels become

entirely obliterated as far back as the superior fundus of the
bladder, and ultimately by a process of progressive atrophy are
converted into fibrous cords, which, resting on the peritoneum,
divide the internal inguinal pouches, each, into an inner and an
outer portion. From the superior fundus of the bladder back-
wards to the edge of the great sacro-sciatic notch, where the
main branches of the internal iliac are given off, the vessels
undergo partial atrophy and diminution of calibre, and under
this form they continue through life as the superior posterior
vesical arteries.

In the horizontal fissure of the liver, the umbilical vein gives
off usually two small branches, which enter and are distributed
in the left lobe; and at the junction of the horizontal and trans-
verse fissures it undergoes ultimate division into two branches;
of these the larger passes in the transverse fissure towards the
right side, to anastomose with the left branch of the *vena portæ*
whilst the second and smaller branch, under the name of *ductus
venosus*, representing the continuation of the parent trunk, runs
upwards and backwards in the horizontal fissure, to open into
the left hepatic vein near the junction of that vessel with the
vena cava ascendens. During fœtal life the intestinal canal is
in a state of functional abeyance, or of preparedness to assume
at birth its proper function. The portal circulation of the fœtus,
therefore, represents only the volume of blood circulated in the
stomach, intestinal canal, spleen, and pancreas, for the purpose of
tissue-nutrition. This, constituting the weaker current, is carried
off by that of the right branch of the umbilical vein in the trans-
verse fissure of the liver, and circulated through the medium of
the right portal branches in the right lobe of that organ. When,
however, alimentary matter is introduced into the stomach after
birth, and the digestive viscera assume, for the first time, their
special function, " a draught," or afflux of arterial blood towards
these organs is the immediate consequence. A full volume of
blood now circulates in the vena portæ, whilst, on the other hand,
the circulation of the umbilical vein has been entirely arrested
by deligation and division of the funis; the current, therefore,
now sets in from the portal to the umbilical branches in the
liver, and, owing to the attraction of secretion operating upon

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the lateral branches, the ductus venosus is entirely drained, and becomes impervious and atrophied, as does likewise the trunk of the umbilical vein from the umbilicus to the transverse fissure of the liver. These vessels are completely obliterated, usually about the fourth day after birth.

Coincidently with the first act of inspiration a series of important organic changes is initiated in the heart. Consequent upon the introduction of atmospheric air into the lungs, an afflux to these organs through the lateral branches of the pulmonary artery takes place, and the ductus arteriosus being entirely and permanently emptied by the draught so established, undergoes gradual obliteration and wasting. The blood which had now for the first time circulated in large volume through the pulmonary vessels, is poured in equal quantity into the left auricle. The operculum is pressed down from the left side upon the foramen ovale, and becomes gradually agglutinated to its margins, closing the passage, as previously described. The entire mass of the blood entering the right auricle is now carried into the right ventricle, the Eustachian valve undergoes gradual wasting, and the head, neck, and upper extremities, no longer enjoying a monopoly of a comparatively pure circulation, as in fœtal life, cease to be developed disproportionately to the rest of the body.

Physiology of the Heart. The *rhythm* of the heart means properly its continued alternate contraction and relaxation at determinate intervals of time; but, as commonly understood, a number of concomitant but subsidiary phenomena, manifest to the senses, is included within the meaning of this term. In this broader sense it may be defined as *the regular and uninterrupted succession, and periodic recurrence, in association with the action of the heart, of certain phenomena of sound and of motion, alternating with brief but unequal intervals of silence and repose, and coeval with the life of the animal.*

The entire group of phenomena, positive and negative, constitute an *action* or *pulsation* of the heart, and of these, within the range of health, and in the sitting posture of the body, from 65 to 95 occur within the minute; the former in advanced age, and the latter in childhood. This standard is arbitrary, but at least approximately correct. The difficulty, universally admit-

ted, of drawing a sharp boundary line between the conterminous regions of health and disease, even structurally in the *post-mortem* room, is greatly enhanced when this has to be accomplished in the living body, and by reference to function exclusively. For groups consisting of great numbers an average may be taken, possessing at least approximate accuracy, but for individuals, owing to the many and various shades of difference in individual organization, each, however, potential in its influence upon the rate of cardiac pulsation, a health-rate cannot be fixed save by reference to the collective standard. I am acquainted with persons in reputedly good health, and who enjoy it in the conventional sense, performing all the duties of their station, involving active and constant labour both physical and mental, whose pulse is habitually not above 50 in the minute. The opposite condition, of a permanently quick pulse, is not equally compatible with apparent health. I am not acquainted with an example, in the adult, of a pulse persistently at 90, in which the existence of disease of some kind was not sufficiently manifest.

The phenomena which constitute a pulsation of the heart, and the succession and repetition of which in regular order, and at determinate intervals, constitute its rhythm, enumerated in the order of their succession from an arbitrary starting point in the cycle, are the following, viz., impulse, wave-like movement more or less localized within the region of the precordium, first sound short pause or period of silence, second sound, long pause.

The regular succession, and unfailing repetition of this cycle of phenomena, constitute one of the most remarkable, as it is the most typical manifestation of animal life. Several theories have been propounded in explanation of the alternate contraction and relaxation of the heart, upon which the above-mentioned sensible phenomena depend. These theories may be classified under two heads, namely, those of *direct*, and those of *mediate* stimulation. Both would imply the presence, in the muscular tissue of the heart, of the property of irritability, in virtue of which it is capable of responding to a stimulus by an act of contraction, the duration of which corresponds with that of the stimulus applied.

Haller[*] was the first writer who systematized the theory of direct stimulation. According to him, the contractility of the heart is quite independent of nervous influence, and brought into action by the contact of the blood with the internal surface of its several chambers. When contact ceases in any particular chamber by departure of the blood from it, contraction of the walls of that chamber likewise ceases, and thus relaxation supervenes; but, owing to the cyclical character of the circulation, and the existence of an auricle and a ventricle on each side of the heart, the former is of necessity full and in diastole, when the latter is empty and in systole. Thus, an alternate contraction and relaxation is maintained, not only in regard to each chamber, but likewise as between the auricle and the ventricle of the same side of the heart.

Nor is this all; for as the circulation is twofold, systemic and pulmonic, the heart is a duplicate organ, and this double alternation of contraction and relaxation is repeated on both sides.

There seems, *in limine,* a fatal objection to this theory, taken as presented by its illustrious author, namely, that it involves the admission of a pre-existing force to set the blood in motion, ere the chambers of the heart could have been excited to contract by contact with it. Or is it that blood having been primarily formed in the heart itself, the property of irritability, by which its presence there was resented on the occasion of the first, as of all subsequent contractions, was, at the outset, *suddenly* conferred upon the organ? This would seem to be a rather gratuitous and unwarranted assumption. Then, again, is the stimulus of black blood required for the right heart, and of red blood for the left? Bichat[†] in his experiments on asphyxia, found black blood circulated freely through the left side of the heart. Finally, there is the objection to this theory, that the heart of a cold-blooded animal, *e.g.,* a frog, may continue to pulsate for a considerable period after its removal from the body of the animal, although during the time no blood has been in circulation through it.

Senac[‡] advocates the Hallerian doctrine in a modified form.

[*] *Opera Minora,* tom. i. p. 155.
[†] *Traité de Physiologie,* par F. A. Longet, 1869, vol. ii. p. 108.
[‡] *Opus citat.,* p. 454.

He maintains that the venæ cavæ and the pulmonary veins contract rhythmically, and propel the blood into the auricles, which, in response to the stimulus of contact, contract upon the blood and force it into the ventricles ; and these, resenting its presence, react and urge it into the aorta and pulmonary artery. He adds, these "machines" continue to act "as long as the vital principle resides in the tissue of the brain and of the nerves." These last words imply a radical modification of the doctrine of "inherent irritability" of Haller.

Brücke* holds that the rhythm of the heart is due to the necessarily intermittent and periodic character of the circulation in the coronary vessels. According to him, the sigmoid valves of the aorta cover the orifices of the coronary arteries, and prevent the entrance of blood into them during the systole of the left ventricle ; but not only is the entrance of blood so prevented, but that already in circulation in the walls of the ventricles is mechanically pressed out through the coronary veins, so that the muscular substance is rendered exsanguine *pro tem.* The effect of this depletion is exhibited in relaxation of the walls of the ventricles, by which blood is permitted to circulate freely through them, and the sigmoid valves being depressed at the same moment, the mouths of the coronary arteries are thereby uncovered, and blood is forced into them by the elastic recoil of the walls of the aorta. The immediate consequence of this is contraction of the ventricular walls, which, by rendering them exsanguine as already explained, gives rise, in turn, to relaxation.

There are many patent objections to this theory. It makes no account of the auricles, which, being likewise supplied with blood by the coronary arteries, and being in diastole when the ventricles are in systole, are therefore in a state of receptivity for blood, when, under the conditions of this theory, none can reach them ; yet, notwithstanding this deprivation, they contract, instead of relaxing, immediately after ventricular systole. Again, immediately succeeding aortic reaction, when, according to Brücke's theory, blood finds entrance into the coronary arteries, and the ventricles should contract, there is a protracted

* Longet (*opus citat*).

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period of silence (the long pause), during which the ventricles are undergoing progressive diastole.

Haller and Bertin, moreover, have observed blood to spout from a punctured coronary artery with increased force during ventricular *systole;* and Hyrtl has recently injected the coronary arteries from one of the pulmonary veins.

I have repeatedly examined the root of the aorta, with a view to determining the relationship between the semilunar valves when raised against the walls of the vessel, and the orifices of the coronary arteries. I have usually found the valves to fall short of the mouths of the coronary arteries by several lines. Occasionally I have seen them touch the lower margin of the orifices, *but in no single instance to cover them.*

The experience of Senac[*] has been similar. He judiciously observes, that blood in some quantity must enter the coronary arteries during ventricular systole, even though the valves when raised, overlay their mouths; because, in being raised, the valves must force into them a portion of the blood resting on their arterial surfaces, whilst it is being pressed out of the sinuses. Morgagni is of the same opinion.

Brown-Séquard has expressed the opinion[†] that the circulation of blood through the coronary veins, charged with *carbonic acid*, constitutes the normal stimulus to the contraction of the heart; and M. Lannelonque,[‡] availing himself of the doctrine of Brown-Séquard, has essayed to explain the difficulty in the way of the reception of Brücke's theory, arising from the alternating contraction of the auricles and the ventricles. According to his view, immediately the ventricles relax, the re-establishment of the coronary venous circulation in their walls, acting as an excitant to the muscular fibres, brings about a renewed act of systole. During auricular diastole, on the contrary, he assumes that circulation in the walls of these chambers is arrested by collapse and " folding " of them; as the auricles fill, the coronary vessels become patulous, and are disgorged only during auricular systole, because, being attached to the muscular fibres,

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[*] *Opus citat.*, p. 365.
[†] *Exper. Researches applied to Physiology and Pathology*, 1853, p. 114.
[‡] *Circulation Veineuse des Parois Auriculaires du Cœur.* Thèse de Paris, 1867.

they are held open by their contraction.   But the assumption of "collapse" or "folding" of the walls of the auricles is not only gratuitous and unproven, but contradicted by observation and experiment, as will appear further on.   The auricles are never empty, because, when disembogued by the divarication of the auriculo-ventricular valves at the moment of cessation of ventricular systole, they are supplied at an equal rate, and with an equal quantity of blood, from the veins opening into them.

Then again, the theory will not hold in reference to the alleged period of disengorgement of the veins, that of auricular systole, because at this period reflux upon the coronary sinus is obviated, either by the closure and adequacy of the valve of Thebesius, or the constriction of the orifices of the vessel by the adjacent contracting fibres.   In either case, the orifice would not be free to the *entrance* of blood during auricular systole.

According to the experiments of Erichsen,* not only the irritability, but likewise the contraction of the heart depends upon the coronary circulation ; he has succeeded in arresting the action of the heart by ligaturing the coronary arteries ; he found, moreover, that this effect was precipitated by puncturing the coronary veins at the same time, and thus rapidly anæmiating the heart-substance, whilst it was postponed when they were ligatured.   Schiff professes to have induced even local paralysis of the heart by tying the artery destined for the supply of that portion of the organ.   Whilst, on the contrary, Ludwig, E. Cyon, and Bezold, deny altogether the alleged influence of deligation of the coronary arteries upon the action of the heart; no immediate consequence of this operation is noticeable, and the changes that subsequently ensue in the number and force of the pulsations, are attributed by them to other causes.

The doctrine of the mediate stimulation of the heart involves the physiology of its nerve-supply, or its *innervation*.   It has been already shown (p. 35) that the heart derives its nerve-supply from two sources, namely, the pneumogastric and cervical sympathetic.   The filaments of these nerves are distributed throughout the substance of the heart, but notably in greatest

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* "On the Influence of the Coronary Circulation on the Action of the Heart " *Lond. Med. Gazette*, 1842.

number on its internal surface, and, in connexion with them minute ganglia have been noticed and described, first by Remak,[*] and subsequently by Bowman,[†] who, however, admits their existence only on the surface of the auricles. From experiments performed by Ludwig[‡] and confirmed by Heidenhain[§] it would seem that these ganglia are located principally in the vicinity of the auriculo-ventricular septa, for when the heart of a frog was divided transversely a short distance below the auriculo-ventricular septum, the auricles and the attached portion of the ventricles continued to pulsate rhythmically for some time, whilst the inferior fragment, separated by section from the ganglionic centre, ceased to contract immediately. Bidder[||] maintains that the coördinating ganglia of the heart are lodged in the inter-auricular septum, and hence the wave-like character of the contractile movement from the auricles towards the ventricles.

To the ganglia situate in the substance of the heart itself, most modern physiologists incline to attribute, as its immediate source, the rhythmical contraction of the organ; but this postulate granted, the precise agency by which the nerve-current emitted from these centres is periodically interrupted, or its influence on the muscle of the heart, whether stimulant or inhibitory, is neutralized at brief and determinate intervals of time, is one of the deepest problems in biology. Mr. Paget,[¶] assuming that the nutrition of the heart is alternately quickened and suspended through the agency of these ganglia, proposes to regard its rhythmical contraction as the consequence of " chronometric nutrition." No doubt, the doctrine which regards nutrition as the remote cause or essential condition of all vital action, upon which Mr. Paget's theory rests, is critically incontrovertible, but this only amounts to presenting the difficulty under a new form, without in any degree diminishing or explaining it.

But the nerve-force, whatever may be its character or effect,

* *Müller's Archiv für. Anat.*, etc., 1844.

† *Physiological Anatomy*, 1856, vol. ii., p. 342.

‡ *Müller's Arch.*, etc., 1848.

§ *Canstatt's Jahresbericht*, etc., 1855.

|| *Müller's Arch.*, 1852.

¶ *Medical Times and Gazette*, January 11, 1859, *Lecture on the Chronometry of Life, and Croonian Lecture at Royal Society*, 1857.

which emanates from the intracordial ganglia, is only borrowed from the cerebro-spinal and sympathetic nerve-centres, through the cardiac branches of the pneumogastric and cervical sympathetic nerves. On the assumption, to be presently discussed, that these nerves are reciprocally antagonistic in function, a few eminent physiologists have recently promulgated the doctrine of alternate stimulation and inhibition, as a solution of the problem involved in the periodicity of the heart's movement.

Notwithstanding the emphatic assertion of Haller* that irritation of the medulla oblongata, spinal cord, or the cardiac nerves, induced no alteration in the action of the heart, physiologists have arrived at the almost unanimous conclusion, that from all three sources a nerve-force emanates, which influences powerfully the rate, the force, and the rhythm of the heart's action. Wilson Philip† was one of the first writers who contributed to the settlement of this question affirmatively, as far as regards the spinal cord. This portion of the nerve-centres of decapitated animals was moistened with alcohol, a solution of opium, and of tobacco, respectively; the first-named agent so applied quickened the action of the heart, whilst the two latter very soon retarded, although at first they accelerated it. He further concluded from his experiments, that the cervical portion of the cord was the most potential in regard to the heart. Prochaska‡ seems to have been the first to assign to the ganglionic nervous system the special function of quickening the contraction of the heart, localizing this function in the cervical, and to a slight extent also, in the superior thoracic ganglia of the sympathetic. Of the independence of this system in regard to its influence over the heart, the examples of amyelencephalic fœtuses§ in which the heart continued to pulsate, notwithstanding the absence of brain and spinal cord, afford striking illustration. In the sequel, however, it will appear, that the evidence thus furnished is by no means conclusive as regards the adult mammal.

* *Dissert. sur l'Irritabilité*, 1756, *traduite de Latin.*
† *An Exper. Inquiry into the Laws of the Vital Functions :* Lon. 1817.
‡ *Opera omnia* (Viennæ, 1800.)
§ *Morgagni, De Sedibus et Causis Morborum, Epistola* xlviii.

Legallois,[*] in 1811, concluded from a series of experiments, that the spinal cord was the source of the contractility of the heart. These experiments were of a character to show that destruction of the lumbar, dorsal, and cervical portions of the cord respectively, in young rabbits, was followed, within a few minutes, by death from failure of the circulation, despite his efforts to sustain it by means of artificial respiration.

The experiments of Legallois were, within a few years, repeated by Wilson Philip,[†] and afterwards by Flourens,[‡] Longet[§] and Brown-Séquard,[||] with entirely contradictory results, as regards the absolute aud exclusive dominion of the spinal cord over the heart. Bernard, moreover, asserts that the results obtained by Legallois were due to the *pain* involved in the operative procedure, and that the previous administration of chloroform would have entirely obviated them.

That electric irritation of the spinal cord, however, is competent to modify in a very decided manner the force, the rate, and the rhythm of the heart's action, the experiments of Volkmann[¶] and Longet,[**] and those of Wilson Philip, already adverted to, fully establish.

Budge and Ed. Weber,[††] in 1845, attributed to certain nerves of the heart the function of restraining its activity. These nerves were named from their alleged function, the *moderator* nerves of the heart.

An influence of this character, under which the heart's action is " slowed," or entirely suspended, the organ remaining in a state of relaxation, may undoubtedly be evoked by stimulation of the medulla oblongata, the pneumogastric nerves, or the abdominal sympathetic; in the two former instances by *direct*, and in the latter by *reflex* irritation.

If the upper part of the medulla oblongata, between the

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[*] *Œuvres Complètes avec des Notes de Pariset*, Paris, 1830.

[†] *Opus citat.*

[‡] *Recher. Expérim. sur les Propr. et les Fonct. du Syst. Nerv.*, Paris, 1824.

[§] *Traité de Physiologie*, 1869, Troisieme edition, tom. ii., p. 113.

[||] *Exper. Researches*, etc.

[¶] *Müller's Archiv.*, 1845.

[**] *Opus citat.*

[††] *Müller's Achiv.*, 1846.

calamus scriptorius and the corpora quadrigemina, be subjected to irritation, the rate of cardiac movement is reduced; and if the irritation be intense, and prolonged beyond a very brief period of time, the heart will cease to contract, and will be found in a state of relaxation. Similar effects will follow from galvanizing the pneumogastric nerves in the neck, and, as shown by Bernstein, the abdominal sympathetic in the frog. If the pneumogastrics be divided in the neck, the pulsations of the heart are promptly increased in frequency, but diminished in force; and stimulation of the medulla oblongata, or of the abdominal sympathetic, will be now without effect upon the heart. If, however, the peripheral portion of the divided pneumogastrics be galvanized, retardation or arrest of the heart's action, according to the intensity of the current, will be the result. It has been already shown that the heart, removed from the body, is capable of sustaining rhythmical action for a lengthened period under favourable circumstances.

It follows, therefore, that the *inhibitory* nerve system of the heart comprises the cerebral origin of the pneumogastric nerves, the pneumogastric nerves themselves, and the ganglia distributed through the heart.

The heart likewise possesses an *excito-motory* system, which consists of the upper part of the spinal cord for centre, and the roots and cardiac branches of the cervical sympathetic for conducting media. If the cervical portion of the spinal cord, or the cervical sympathetic, be galvanized, the heart will pulsate with increased rapidity and force,

Ludwig, however, maintains, that the only portion of the cervical sympathetic which is related to the heart is the inferior cervical ganglion, inasmuch as stimulation of it alone has any effect upon the heart. Dr. Rutherford is of the same opinion; and after discussing very fully the entire subject of the innervation of the heart, this writer summarizes his views in the form of propositions, as follows:

1. The inferior cardiac branches of the vagi are inhibitory of the heart, and in no sense motor, as held by Willis, Lower, Valsalva, Schiff, Moleschott, and Lister.

2. They are not in constant action; activity is the exception.

3. Increase in the rapidity of cardiac movements which follows division of the vagi in the neck, may be due to increase of blood-pressure merely, and this latter to contraction of the gastric blood vessels (owing to suspension of the reflex "depressory" function of the vagi upon the abdominal vessels.

4. The contractile coats of the entire vascular system are supplied with *motor* and *inhibitory* nerves.

5. During digestion the blood vessels of the stomach are dilated, chiefly by vaso-inhibitory action of the vagi upon the splanchnic nerves.*

These two systems, the inhibitory, and the excito-motory, are connected with one another *centrally* through the spinal cord and medulla oblongata, and *peripherally* through the cardiac plexuses and the ganglia distributed through the substance of the heart.

The necessity for the existence of two opposing and mutually countervailing systems of nerves in the heart, would seem to be involved in the *promptitude* with which, in carrying on the circulation, it is required to contract and relax alternately.

It is alleged that the rapid and continued succession of powerful motory nerve-impulses to which the heart is subjected, would, as in the case of voluntary muscle similarly treated, give rise to persistent spasm, or tetanic contraction of the organ, a condition incompatible with the maintenance of the circulation. An "inhibitory" nerve system, however, which operates, not in the Hunterian sense, by inducing a state of "active relaxation" of the heart, but by neutralizing or suspending, *pro tem*, the motor influence directed upon it from and through the excito-motory system, and which, in turn, either by temporary suspension or relative diminution in the force of its nerve-current, is controlled or overpowered by that system, is manifestly competent to supply the conditions necessary for the maintenance of the circulation, under the special circumstances indicated.

Longet † asserts that the necessity for temporary "inhibition" of the heart has reference to the repose and nutrition of the

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* *Journal of Anatomy and Physiology*, No. iv., May, 1869.
† *Opus citat.*, p. 120.

organ. This is manifestly true; but a necessity no less urgent, requiring the temporary and alternate suspension of contraction in each of the chambers of the heart, has reference to the maintenance of the general circulation. It is manifest that the distension of each of those chambers with a fresh charge of blood, preparatory to a new act of contraction, implies an antecedent state of relaxation of its walls.

Division of the pneumogastric nerves in the neck gives rise to derangement of the normal ratio between respiration and cardiac pulsation. The former is " slowed " by one half, whilst the latter is doubled according to Bernard. Inspiration is full and deep, yet the blood is imperfectly arterialized, and notwithstanding the excited action of the heart, arterial tension is notably reduced.

Bernard remarks, as a singular anomaly, that in the dog, the pulse, which is normally irregular, is rendered perfectly regular by section of the pneumogastrics. In the frog, on the other hand, Brown-Séquard alleges that division of these nerves has no influence whatever upon respiration or cardiac pulsation.

Galvanization of the pneumogastrics, or of the peripheral extremity of the divided nerves, will cause arrest of the heart in a state of diastole. For this purpose a strong current is necessary; but if too powerful, or long continued, it will have the opposite effect of accelerating the action of the heart, like section or ligature of the nerves. In this case, likewise, the force of the heart will be lessened, as evidenced by diminished arterial tension. When the current has been stopped, the pulsations return gradually to their normal state. Schiff asserts that galvanization of a single nerve, if sufficiently energetic, will suffice to produce the effects above mentioned.

The hypothesis of " nervous exhaustion " has been adduced, singularly enough, by different physiologists, to explain both the quickening and the arrest of the heart's pulsations, which result respectively from section and galvanization of the pneumogastric nerves in the neck. It is manifest that one of these doctrines must be erroneous. Schiff, Spiegelberg, G. Valentin, Jos. Lister, and Moleschott, hold that the arrest of the heart's action is due to this cause; but this doctrine would imply that

the pneumogastrics act as motor nerves upon the heart, a position utterly untenable, because irreconcilable with the admitted fact, that whilst a moderately strong current stops the action of the heart, a very strong, or a moderate but prolonged current, will induce rapid but feeble pulsation. The last-mentioned result is presumably due to exhaustion, but if so, the former, which is precisely the reverse, cannot be in any degree attributable to the same cause.

Curara poison previously administered, by annihilating the excitability of the heart, will neutralize the effect of a galvanic current applied to the pneumogastic nerves. Digitalis in large doses will arrest the heart in a state of diastole; its effect upon the heart is therefore similar to that of galvanization of the pneumogastic or its central nucleus; and that it acts upon the heart through the same channel was proved by the experiment of Traube, which showed that it fails to act where the pneumogastrics have been previously divided.

Waller* alleges that the "inhibitory" property of the pneumogastric nerves is derived from the spinal accessory. He found that when, after destruction of the spinal accessory of one side, the divided roots had undergone fatty degeneration, galvanization of the corresponding pneumogastric failed to cause arrest of the heart, and Longet† has shown experimentally that a motor nerve, detached from the cerebro-spinal centre, ceases to be excitable after the fourth day.

The question of the innervation of the heart was, in 1863, still in a state of uncertainty, to the extent that it was not settled, on the one hand, whether the heart is entirely independent of the spinal cord, and on the other, whether the central nervous system can influence the heart, save through the pneumogastric nerves; the experiments and conclusions previously adverted to, notwithstanding.

In that year (1863) Professor Bezold concluded from the following experiment, that he had proved the existence in the spinal cord of an excito-motory centre of the heart, which was

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* *Expérience sur les Nerfs Pneumogastriques et Accessoires de Willis* (*Gazette Medicale de Paris*, 1856.

† *Recherches Experimen.* (*Examinateur Med.* 1841).

capable, not only of increasing the number of its pulsations, but likewise of augmenting considerably the mean pressure of the blood; section of the cord at the level of the atlas in rabbits reduced the pressure in the large arteries, and slowed the heart. Irritation of the cord below the level of section increased both the vascular pressure and the number of cardiac pulsations. These results he attributed exclusively to diminution or augmentation of force of contraction of the heart. MM. Ludwig and Thiry found the same results to follow these experiments, after destroying all the nerves of the heart by the galvanocaustic method, and that pressure on the abdominal aorta caused equal acceleration of heart and increase of vascular pressure; they arrived at the seemingly warrantable conclusion, that the results obtained were due, in the one case to paralysis, and in the other to irritation of the vaso-motor nerves, which, by causing diminution or increase of vascular resistance to the heart, reduced the pressure and rate of pulsation, or increased both.

Ludwig and E. Cyon, by their conjoint experiments, have established; firstly, that the cardiac nerve arising by two roots from the pneumogastric and the superior laryngeal, is a sentient nerve of the heart, conferring on it the power of regulating vascular pressure throughout the body, by inducing, through reflex influence, paresis of the vaso-motor nerves, and so temporary paralysis of the walls of the blood-vessels: to this nerve they therefore propose to give the name of *depressor nerve.* And secondly, that the splanchnic nerves are the principal vascular nerves of the body, section of them reducing pressure in the carotids to a minimum, and irritation of the distal ends of the divided nerves doubling it.

In the hope of excluding, by section of the splanchnics, all vascular changes during irritation of the spinal cord, the brothers Cyon* performed the following experiments: Rabbits were poisoned with curara, and artificial respiration was kept up; the pneumogastric, the sympathetic, and the "depressor" nerves were next divided in the neck, and then the number of beats of the

* *Sur L'Innervation du Cœur par MM. E et M. Cyon, de Saint Pétersbourg* (note transmise par M. Cl. Bernard, *Comptes Rendus de L'Académie des Sciences,* lxiv, Mars, 1867.

heart was registered, and by means of the manometer of Ludwig the blood-pressure in the carotids was determined, *before, during,* and *after* electric irritation of the spinal cord, separated from the brain at the level of the atlas. After having ascertained a considerable augmentation of blood-pressure, and an increase in the number of cardiac pulsations, consequent upon irritation of the spinal cord, they divided the two splanchnics below the diaphragm, and quickly the blood-pressure, and the number of pulsations of the heart, descended even below the standard attained after division of the spinal cord *only.* Irritation of the cord, after division of the splanchnic nerves, produced again a considerable acceleration of the action of the heart, *but no alteration of blood-pressure.* From these experiments it may be inferred that spinal irritation causes accelerated pulsation of the heart through the cardiac nerves; and increased vascular pressure through the splanchnics, by means of the vascular resistance induced.

The " depressor " nerve was divided, and the peripheral extremity galvanized without result. The central end of the divided nerve was next subjected to galvanic irritation; the animal immediately gave evidence of pain, and there was a fall of five to six centimetres in arterial tension. These phenomena ceased immediately the current was suspended. If the cardiac nerves were all previously divided, the above-mentioned consequences nevertheless followed; but if the splanchnics were divided they promptly ceased to be manifested.

These experiments prove that the " depressor " nerves are centripetal; and that they exercise their reflex influence, not immediately through the cardiac nerves upon the heart, but on the blood-vessels of the body through the vaso-motor system, of which the splanchnics are the great centre; and finally, that this influence is of a paralyzing or paresic character, causing relaxation of the vascular walls, dilation of the vessels, and consequent subsidence of arterial tension.

All the branches which the heart receives from the spinal cord, through the medium of the lower cervical and upper dorsal ganglia, were then extirpated in other rabbits; the splanchnics were divided, and the cord irritated as before, but no change occurred, either in the number of cardiac pulsations, or in the

mean intra-vascular pressure; thus confirming the preceding inference. It was noted, however, that when the irritation was protracted, an elevation of two to three millimetres in the vascular pressure took place, which was attributed, with some probability, to irritation of the vascular nerves arising lower down than the splanchnics. It was found, moreover, that extirpation of the spinal roots of the cardiac nerves *alone* produced no change either in the number or value of the contractions of the heart. From this the experimenters conclude, I think unwarrantably, that "these nerves do not act in a continuous manner." In my opinion it rather shows, that whilst these nerves constitute the ordinary channels for the conduction of positive irritation from the spinal cord to the heart, the latter organ may discharge, temporarily at least, its ordinary functions independently of them, obtaining its supply of nerve-force from its intrinsic ganglia.

Electric irritation of the third branch of the inferior cervical ganglion provoked, in rabbits, an acceleration of the beats of the heart, and a diminution of their length; and the first and second branches of the same ganglion were found to be sentient nerves of the heart, and to constitute a continuation of the "depressor" nerve.

Irritation of the fourth branch of the same ganglion, which passes in front of the subclavian artery, and forms with the fifth the annulus of Vieussens, produced a slight elevation of blood-pressure without changing the number of the pulsations.

The *direct* irritation of these cardiac nerves, in dogs and rabbits, produces less acceleration of the heart's action than *indirect* irritation through the spinal cord, because, in the latter case, *all* the cardiac nerves are simultaneously irritated.

The third branches of the inferior cervical ganglia, they propose, therefore, to designate the *accelerator* nerves of the heart. They are not ordinary motor nerves terminating in the muscle of the heart, because irritation of them will not give rise to tetanic contraction of that organ, nor increase the sum of work done by it, as shown by the fact determined by means of the manometer, that whilst the number of pulsations was increased, their force was lessened; and also because curara will not para-

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lyse them. Neither are they the nerves which act upon the nutrient vessels of the heart, because occlusion of these vessels will not give rise to acceleration of the heart's action.

The conclusions arrived at in regard to the termination and special function of the "accelerator" nerves are, that they end in the ganglia of the heart; that they are subject to the reflex influence of a great number of the sentient nerves of the body, but especially those distributed on the internal surface of the heart; that their action consists in effecting a change of the division *in time* of the heart's work; and finally, that they are in antagonism to the pneumogastrics in this sense, that whilst irritation of the latter "slows" the heart but increases the force of its contractions, irritation of the "accelerator" nerves increases the number of the pulsations, but diminishes their force.

Brown-Séquard* is of opinion that the pneumogastrics are the vaso-motor nerves of the heart, and that their "inhibitory" influence upon that organ is the result of contraction of the coronary vessels, and temporary anæmia of the walls of the heart. But this view of the function of the cardiac branches of the pneumogastrics would imply, that under their active influence the walls of the heart are in a state of anæmia, which is not the case; and that, as in the experiments of Erichsen and Schiff, of ligaturing the coronary arteries, the relaxation of the heart would be slow and gradual, and the arrest of its action complete only after an interval of several seconds consecutive to the operation; not sudden and prompt as in the experiment of galvanizing the pneumogastric.

That the ganglia of the heart possess, and may exercise for a considerable period independently of the nerve centres, a power of influencing the movements of the heart; and that this power is regulated by, or dependent upon, the blood-supply furnished by the coronary arteries, seems proved by the following experiment of Bezold.† When he closed the trunks of the coronary arteries or several of their branches, by means of forceps, after having divided the spinal cord, the vagi, and the sympathetic nerves in the neck, he observed that during the first ten or

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* *Gazette Med. de Paris*, 1853.

† Quoted by Eulénberg, *Medical Times and Gazette* of 23rd April, 1870, p. 433.

fifteen seconds no perceptible change took place; then the heart's beats became slow and irregular, half or three quarters of a minute intervening; and after a minute or a minute and a-half the ventricles ceased to act. But when the obstruction was removed, pulsation began again, and soon became perfectly regular.

From what has preceded, it will be evident that the special and distinctive functions of the several nerves of the heart have not been yet clearly determined.

After an excellent *résumé* of this subject, Longet[*] remarks, " We admit, nevertheless, that new experimental researches are necessary, in order to determine the precise laws of the innervation of the heart."

Convinced of the truth of this statement, and no less, of the importance of the subject in regard to the localization and treatment of certain symptoms, not only of cardiac, but of febrile disease, I determined, in conjunction with my young friend Dr. Furlong, whose information and special aptitude for investigations of this kind qualify him in a high degree for the task, to undertake a new and independent series of experiments, with the view of still further elucidating it, and of endeavouring especially to settle the question of the precise and special role of the individual nerves in the physiology of the heart. I regret to say, however, that after Dr. Furlong had made all the preliminary arrangements, devised and constructed instruments, procured a supply of curara, and received from the Royal Irish Academy a grant in furtherance of his experimental inquiry,[†] the investigation was abruptly brought to an end by his having been called to an extensive practice in the country,[‡] which absorbed his whole time. I thus, to my great regret, found myself without a collaborateur, and unable to command the time necessary to pursue the investigation single handed. I sincerely hope that some physiologist, younger, more vigorous, and in other respects better qualified for the task than I am, may undertake and accomplish it.

[*] *Opus citat*, p 124, foot note.

[†] A preliminary Report, together with the instrument devised for the purpose of carrying on artificial respiration, was submitted to the Royal Irish Academy in the session 1872-3. *Vide Report of Proceedings*.

[‡] Enniscorthy.

Having discussed somewhat fully the anatomy of the heart generally, and its nervous endowment in particular, I may now enter upon the consideration of its physiology. A clear conception of the mechanism of the heart; of the agency by which its movements, and those of its several parts, are initiated and controlled; of the coincidence or the asynchronism of its many sensible phenomena; and of the character, quality, and duration of each of these in the state of health, is absolutely necessary as a first step in the acquisition of a knowledge of the deviations which indicate derangement of its function; the source whence they proceed; the seat of their origin; the cause to which they are due; their diagnostic and prognostic significance; and the therapeutic indications arising therefrom. I am guilty of no exaggeration in asserting that upon this subject, even in the minds of otherwise accomplished physicians, the utmost confusion has existed, and still, but in a much less degree, exists.

The substitution of accurate knowledge for hasty assumption and adventurous hypothesis, upon a matter of so much importance, should, therefore, be the first object of any one undertaking to produce a treatise on the Diseases of the Heart, on a level with the present advanced knowledge of its physiology.

It has seemed to me that much useful information may be afforded by presenting, in a synoptical form, the principal hypotheses hitherto propounded in regard to the rhythm of the heart, and the grouping or succession of its several phenomena. But as these are presented in a cycle, which, throughout life, is being constantly repeated, without material deviation in health, but exhibiting in disease various alterations of corresponding significance, the diagramatic scheme of a circle seemed best adapted to the object in view.

In the construction of this scheme the cardinal point to be attended to was the contraction of the ventricles, because with it were associated in the minds of all writers on the subject, with only three notable exceptions, and upon it depend, the leading phenomena of impulse and first sound. The relaxation of the ventricles possesses only a reflected value, and constitutes the second point of importance to be attended to.

But these two principal phenomena were found to cover the

entire period of a cardiac cycle, and therefore all the minor phe-
nomena were readily grouped in two great divisions under them.

Lancisi (Fig. I.) divided the cycle into two equal portions,
namely, ventricular systole, and ventricular diastole. Within
the former were grouped the impulse of the heart, the first
sound, the two posterior thirds of the auricular systole (coinci-
ding with the two anterior thirds of ventricular systole), closure
of the auriculo-ventricular, and opening of the arterial valves,
escape of blood from the ventricles into the aorta and pulmonary
artery, and distension of these vessels. Within the period of
ventricular diastole occurred the second sound, the anterior third
of auricular systole (coinciding with the terminal portion of ven-
tricular diastole), opening of auriculo-ventricular, and closure of
arterial valves, and reaction of the aorta and pulmonary artery.
The subjoined diagram will render this intelligible and easy to
remember.

Fig. I.

a	*	...	.	. .	*	Ventricular systole.
b	*	...	...	...	*	Ventricular diastole.
c	*	...	*	...	*	Auricular systole.
d	*	. .	*	...	*	Auricular diastole.

The manifest objection to this scheme is that it assumes,

Fig. II

a *   — —   .   *   Ventricular systole (*primum tempus*).
b *   .   *   Ventricular diastole (*secundum tempus*)

contrary to all mechanical principles, and to observed facts, that the contraction and the dilatation of the auricles and of the ventricles respectively, are in greatest part simultaneous. It may be, therefore, set aside without further discussion; but historically it is worthy of being recorded as evidence of the importance with which, even at that early period,* a concrete knowledge of the rhythm of the heart was invested in the mind of a great physiologist and physician.

Haller (Fig. II.) divided the entire cycle into two equal portions, corresponding respectively to what he designates *primum tempus*, and *secundum tempus* Within the former period occur the following phenomena, viz.: contraction of the ventricles, impulse, first sound, displacement of anterior or larger segments of auriculo-ventricular valves from arterial orifices, and closure of these valves over the corresponding orifices, with repulsion into auricles of a " cone of blood," raising of arterial valves, and distension of aorta, pulmonary artery, and auricles.

* First decade of seventeenth century.

During the latter period (*secundum tempus*) occur the relaxation of the ventricles, and entrance of blood from the auricles, overlapping of arterial orifices by anterior segments of auriculo-ventricular valves, reaction of aorta and pulmonary artery, closure of arterial valves, second sound, and contraction of auricles.

This sketch is in greatest part correct, so far as it extends; the most notable exception being, that the auriculo-ventricular valves are floated athwart the corresponding orifices by the blood entering from the auricles, instead of being forcibly raised, and lifting a column of blood into the auricles, under the contraction of the ventricles, as implied by Haller. There is, moreover, a want of fulness of detail, and a total omission of several features essential to a complete scheme, such as an apportionment of time.

Laennec regarded the sounds as the principal phenomena, attributing the first sound to the contraction of the ventricles, and the second sound to that of the auricles. The error implied in this assumption, as well as that of the relative apportionment of the entire period of the cycle, exhibited in the next figure (Fig. III.), requires no explanation.

Fig. III.

a First sound (Ventricular systole) = $\frac{1}{2}$ of cycle.
b Second sound (Auricular systole) = $\frac{1}{4}$ of cycle.
c Interval of repose of entire organ = $\frac{1}{4}$ of cycle.

He held that there was no "interval of repose" between the first and second sound; but herein he was in error, as he likewise was in the assumption, that every twenty-four hours the ventricles enjoyed twelve, and the auricles eighteen hours of repose. If, by diastole, is meant ventricular repose, then the ventricles actually enjoy sixteen hours of such out of every twenty-four. But diastole is not repose or immotion; it is relaxation and passive motion, and not entirely exempt from tissue-waste. The auricles, as will be hereafter pointed out, are in action during the entire period of ventricular diastole, i.e., during two-thirds of the period occupied by a cardiac cycle; but in vigorous action only in the terminal portion of this period. Their state of relaxation corresponds to ventricular systole, i.e., one-third of the cycle; but during ventricular systole the auricles are in a state of extreme distension, which cannot be regarded as repose. With the first sound, according to Laennec, the impulse of the heart and the radial pulse coincide.

Bouillaud (Fig. IV.) divided the period of cardiac action into two equal parts, viz., ventricular systole, and ventricular diastole.

During the systole occur, the impulse of the apex; first sound; recession of the body of the ventricles; pulsation of adjacent arteries; repose of auricles and suction of blood into them; contraction of papillary muscles and consequent closure of auriculo-ventricular valves; and efflux of blood into the aorta and pulmonary artery.

Within the period of diastole occur, the retrocession of the apex from the chest-wall; the second sound; slight retraction of auricles; repose and partial distension of ventricles; vermicular contraction of auricles, commencing in appendices and ending in ventricles, where it merges in ventricular systole; reaction of aorta and pulmonary artery, and consequent closure of sigmoid valves, after being drawn towards base by ventricular diastole.

In reference to this scheme I will merely observe, that the retreat of the body of the ventricles from the anterior thoracic wall after the impulse, takes place, not within the period of ventricular systole, but during the subsequent diastole; and that no notice is taken of the energetic contraction of the auricles, which

Fig. IV.

a Ventricular systole = ½ cycle.
b Ventricular diastole = ½ cycle.

occurs immediately before the contraction of the ventricles, upon which the latter depends, and to which, as will be shown in the sequel, the pathognomonic murmur of mitral constriction is mainly due.

The Dublin Committee of the British Association (1835) divided the entire period of the heart's action into three unequal portions (Fig V.); one-half corresponding to the ventricular systole, the impulse, and the first sound, which are all of equal length; one-eighth belongs to the second sound; and three-eighths to the subsequent pause, during which diastole of the ventricles, and systole of the auricular appendices occur. The first sound is caused by *bruit musculaire*, and rushing of blood over the rough surface of the ventricles; it is intensified by impulse against the wall of the chest. The second sound is due exclusively to closure and tension of the sigmoid valves. This scheme, though possessing much merit, and being decidedly the best up to that date produced, because based upon direct observation and ex-

periment, was erroneous in assuming that the diastole of the auricles does not occur till after the lapse of an appreciable interval subsequent to the systole of the ventricles, whereas it actually coincides with the latter. It was also at fault in excluding the second sound from the period of ventricular diastole; and in representing the auricular appendices only as contracting during the pause. The division of time was likewise incorrect; and the impulse and first sound were both erroneously assumed to be equal in length to the systole of the ventricles.

The following summary of the conclusions arrived at by the Dublin Committee are eminently worthy of attention, as indicating the great acumen of the observers constituting that distinguished body, and as presenting the least incorrect view of the action of the heart propounded up to that date:

1. " In warm-blooded animals the systole of the ventricles follows immediately that of the auricular appendices.
2. " During the systole of the ventricles the auricles are distended by blood from the venous trunks.

Fig V

a First sound (ventricular systole and impulse) . ... .. $= \frac{1}{4}$ cycle.
b Second sound ... ... $= \frac{1}{4}$ cycle.
c Pause (ventricular diastole, and systole of auricular appendices) $= \frac{1}{2}$ cycle.

3. "When the systole has ended, the ventricles become re-laxed and flaccid, and blood passes rapidly, but not with force, from the auricles into their cavities.

4. "The auricles are never emptied of their blood, and contract but little on their contents, an active contraction being observable only in their appendices.

5. "If the interval between two successive beats of the heart be regarded as divided into four equal parts, two of these may be allotted to the duration of the ventricular systole, something less than one to the interval between the termination of the ventricular systole, and the beginning of the diastole of the appendices, during which interval little motion is observed in the auricles; and the remainder to the diastole, and systole of the auriclar appendices.

6. "The ventricles in their systole approach the front of the thorax, and by their contact and pressure against it, produce the impulse or beat of the heart."

Corrigan divided the cycle into three unequal parts (Fig. VI.), regarding the sounds as the principal events, after the example of the Dublin Committee. The first sound occupied one half the cycle, and coincided with the impulse of the heart, the contraction of the auricles, and dilatation of the ventricles. The second sound extended over one-fourth of the cycle, and coincided with the contraction of the ventricles, the distension of the aorta and pulmonary artery, and the radial pulse. The pause engaged likewise one-fourth of the cycle, and corresponded to the distension of the auricles and relaxation of the ventricles.

This scheme is eminently heterodox, and has been recently repudiated by its eminent author, who, on witnessing some conclusive experiments by M. Chauveau,* is understood to have announced his conversion.

Long anterior, however, to the date of Chauveau's exposition, he had practically abandoned it, and conformed to the orthodox view, as is well known to his professional brethren in Dublin. Nevertheless, as no formal retractation has been published, it is necessary to state briefly the objections to this scheme.

The first sound was made to coincide with the contraction of the auricles, and dilatation of the ventricles. The second sound

* *Dublin Hospital Gazette* November 1, 1860.

was represented as coinciding with the contraction of the ventricles and the radial pulse; and from the pause was excluded

FIG VI

a First sound (contract. of auricles, dilatation of ventricles, and impulse) — ½ cycle.
b Second sound (contract. of ventricles, and radial pulse) ..   .. — ¼ cycle.
c Pause (distension of auricles, and relaxation of ventricles) ..   . .. — ¼ cycle.

the contraction of the auricles. The radial pulse could never, in this view, be anterior to the second sound, since ventricular systole on which it depends, coincides with that sound; yet it is well known to be normally antecedent to it. M. Beau takes a similar view as to the cause of the sounds of the heart, and is, therefore, likewise amenable to the foregoing strictures.

Corrigan adverted to "a mistake made by Laennec, who stated that the ventricles contracted before the auricles. It was now generally agreed that Laennec was wrong."* I fail to perceive the force of this remark, since any one point of a circle may occupy a place before or after any other point in it, according to the point of departure selected.

Carlile divided the cycle into three parts (Fig. VII.); the first, occupying three-eighths, corresponds to the first sound, the sys-

* British Association, 1835.

Fig. VII.

a First sound (v. systole) ... — ⅜ cycle
b Second sound ... . = ⅛ cycle.
c Pause ... . = ½ cycle.

tole of the ventricles and closure of the auriculo-ventricular valves, impulse of the heart, distension of the aorta and pulmonary artery, and the diastole of the auricles. The second part represents the second sound, and is one-eighth of the whole cycle; it corresponds likewise to the reaction of the aorta and pulmonary artery, closure of the sigmoid valves, relaxation of the ventricles, opening of the auriculo-ventricular valves, and entrance of some blood from the auricles. The third part, amounting to one-half of the cycle, represents the pause, and corresponds to the systole of the auricles, which commences at the entrance of the venæ cavæ and ends at the appendix; the diastole of the ventricles, and entrance of the principal volume of blood from the auricles.

This was the most truthful and accurate sketch given up to the date of its publication; still, several objections apply to it. The time is incorrectly apportioned, the sounds are made the principal events, and the systole of the auricles is placed at the commencement, instead of the termination of the long pause.

He attributed the first sound to the rushing of blood through the aortic and pulmonic orifices.

Hope implies a division of the cycle into two equal parts (Fig. VIII.) The first portion is appropriated by the ventricular systolic phenomena, viz.: first sound, impulse, and passage of blood into the great arterial trunks. To the last-mentioned phenomenon he attributes the first sound. The second period is occupied by the ventricular diastolic phenomena, viz : the second sound, and the passage of blood from the auricles into the ventricles, to which that sound is due.

This is a most defective scheme, and erroneous *quoad* the causes to which the sounds of the heart are attributed.

Gendrin furnishes a scheme of great complexity (Fig. IX.), but possessing some special merits. It is, however, in many particulars erroneous. In it the cycle is divided into six parts: viz., *systole*, equal to one-fourth of cycle; *perisystole, prediastole*, and *diastole*, together corresponding to one-fourth; *peridiastole*, and *présystole*, together equal to one-half. Within the

FIG. VIII

a Ventricular systole, etc   ...   . = ¼ cycle.
b Ventricular diastole, etc. ...   = ¼ cycle.

period of systole occur, the contraction of the ventricles, first sound, impulse, efflux from ventricles into great arteries, initial diastole of latter, and vibration of corpuscles of blood. The perisystolic phenomena are, incipient relaxation of ventricles, and closure of arterial valves. In the prediastole the preceding phenomena are continued.

Fig IX.

a Systole ... . — ¼ cycle.
b Perisystole ⎫
c Prediastole ⎬ ... ... = ½ cycle.
d Diastole ⎭
e Peridiastole ⎫ ... ... = ¼ cycle.
f Presystole ⎭

The diastole comprises the relaxation of the ventricles and auricles; opening of auriculo-ventricular valves, and entrance of blood from great veins into auricles, and from auricles into ventricles; recoil of this blood from apex against base and closed arterial valves; second sound.

Peridiastolic phenomena are, complete relaxation of auricles and ventricles, and free entrance of blood from great veins into auricles, and from auricles into ventricles; and during presystole, contraction of the auricles and rapid entrance of blood into ventricles take place.

The first sound Gendrin attributes to the vibration of the corpuscles of the blood; and the second sound, to the recoil of the blood first entering the ventricles, from the apex, against the base of the heart and the closed arterial valves. He holds that the closure of the arterial valves precedes the second sound.

The three events, however, are really simultaneous; namely, closure of the sigmoid valves, the second sound, and the first entrance of blood into the ventricles; and, as is now well established, the second sound is caused by the abrupt closure and tension of the sigmoid valves. He alleges that the second sound exists after the sigmoid valves have been destroyed. This is an error, as proved by experiment on living animals, and likewise by the total absence of second sound in the aorta when the sigmoid valves are so much disorganized as to be entirely incompetent.

M. A. Luton,[*] copying the scheme of Marey and Chauveau

Fig. X

a Period of activity   ..   = ⅓ cycle.
b First stage   ...   .   = ⅕ cycle.
c Second stage   ..   ..   = ½₀ cycle.
d Period of repose (third stage) . — ½ cycle.

[*] *Nouveau Dictionnaire de Médecine et de Chirurgie Pratique*, Paris, 1868.

as the result of their experiments upon the horse, divides the cycle into two principal and equal portions (Fig. X,, viz., a "period of activity" and a "period of repose." The former includes two stages; the first stage comprising the contraction of the auricles, and the active diastole of the ventricles. This stage is equal to one-tenth of the cycle, or of one second, the rate of the pulse being sixty per minute. In the second stage of this period, which extends over four-tenths of a second, the pulse rate being sixty, occur, the contraction of the ventricles and the impulse, which are of equal duration; first sound; closure of auriculo-ventricular valves, and passage of blood into aorta and pulmonary artery.

The "period of repose," pulse being at sixty, equals five-tenths of a second, or of the whole cycle, and corresponds to

Fig. XI.

a Ventricular systole.
b Ventricular diastole.
c Rest.
d Auricular systole.
e Arterial pulsation in neck.
f   ,,   ,,   in wrist.
g   ,,   ,,   in foot.

6*

the third stage of cardiac movement. In it are included the passive diastole of the auricles and ventricles; closure of sigmoid valves, and second sound.

The great eminence of the physiologists who are the authors of this scheme entitle it to a ready and favourable reception, irrespectively of its intrinsic merits, which are very considerable. In this sketch of their opinions, however, there are some cardinal errors, which, as will be shown further on, have been since eliminated from their scheme.

Gairdner (Fig XI.) divides the cycle into three nearly equal portions; viz., ventricular systole, ventricular diastole, and rest. The period of ventricular systole includes the first sound, cardiac impulse, and arterial pulsation in neck, wrist, and foot, in succession. Ventricular diastole embraces the second sound; whilst rest is neutral as regards sensible phenomena, but includes, at its termination, the systole of the auricles. This scheme possesses many and great merits, prominent amongst which is the recognition, for the first time made in diagrammatic form, of the true

FIG. XII.

a First sound, V. systole, A. diastole * ... *
b Ventricular diastole ... ... * .. *

relative position of auricular systole. The apportionment of time as represented, is, however, notably incorrect.

Dr Markham (Fig XII.) represents the cycle as divisible into three unequal portions, viz, that of the first sound, amounting to about two-fifths of the whole, during which, and nearly synchronous with it, the impulse of the heart, the pulsation of the arteries in the neck and wrists, the systole of the ventricles, and the diastole of the auricles take place : that of the second sound, corresponding to the commencement of ventricular diastole, and the recession of the apex after the impulse, and occupying somewhat less than one-fifth of the cycle; and finally the pause, extending over somewhat more than two-fifths, or about one-half the cycle, and during which the completion of ventricular diastole and auricular systole take place.

The ventricular systole, the first sound, and the auricular diastole, although synchronous, are not coeval; that is, they com-

Fig XIII.

a First sound = ¼ cycle.
b Second sound — ⅓ cycle.
c Pause .   — ½ cycle.

mence but do not terminate at the same time. The systole of the ventricles and the diastole of the auricles cover the entire period, whereas the first sound occupies only the initial portion of it. The foregoing objections are applicable to this system, irrespectively of the mal-apportionment of time which may be likewise urged against it; but it has the great merit of having been the first promulgated in the English language in which an accurate appreciation of the rhythm of auricular systole is indicated.

Dr. Pavy's scheme (Fig. XIII.) consists in a division of the cycle into three parts, viz., that of the first sound, that of the second sound, and that of the pause. Each of the two former occupies one-fourth, and the latter one-half the cycle. Within the period of the first sound may be noted the impulse of the auricles; the contraction of the ventricles; tension of auriculo-ventricular valves; distension of the aorta and pulmonary artery; tilting forwards and to the right side, of the apex; and the

Fig. XIV.

a First sound	.. ...	= ¼ cycle.
b Second sound	.. ...	= ¼ cycle.
c Pause	.. ...	= ½ cycle.

impulse of the heart. During the period of the second sound reaction of the aorta and pulmonary artery occurs; flapping down of the semilunar valves; contraction-wave of auricles towards ventricles; incipient relaxation of, and entrance of blood into, ventricles. The period of pause corresponds to the complete relaxation and gradual distension of the ventricles, and the closure of the auriculo-ventricular valves.

The relative proportion of time assigned to the sounds in this scheme is obviously incorrect, and the active contraction of the auricles at the end of the pause is not recognized.

Halford (Fig. XIV.) likewise adopts the tripartite division of the cycle, assigning, however, to the first sound one-half, and to the second sound and the pause, one-fourth respectively.

This scheme is open to the objections urged against that last described; and further, the author does not admit a short pause. According to him, the apex of the heart does not strike the wall of the chest in ventricular systole; it is pressed downwards and backwards, and from right to left, and the impulse at the ribs is most probably given by the fibres just above the apex. He holds that the auricles continue "filling after the second sound is heard." This can only mean that blood continues to flow into them from the cavæ, coronary, and pulmonary veins, because, the auriculo-ventricular valves being open, it most probably flows as quickly from, as into, the auricles. He considers that the blow or impulse of ventricular systole is due to "pressure of the opposing column of blood on the upper surface of the semilunar valves." The pressure alluded to must be comparatively insignificant, as it is shown to be in Marey's tracings. The volume of blood sent out by the preceding contraction of the ventricles has already circulated through the pulmonary and systemic vessels; the reaction of the arteries has long since taken place, and been succeeded by the protracted period of arterial quiescence corresponding to the long pause. The pressure on the sigmoid valves by the opposing column of blood, at the moment of ventricular systole, must therefore be quite inadequate to explain, on the principle of collision of opposing forces, the systolic impulse of the ventricles. With Billing, Rouannet, Bryan, Valentin, Baumgarten, and Brakyn, he holds that the first sound is entirely

Fig. XV.

a Ventricular systole and first sound   ...   ...   = ⅓ cycle.
b Second sound   ...   .   = ⅙ cycle.
c Ventricular diastole   < ....> ...   = ⅔ cycle.

valvular; and more prolonged than the second, owing to vibration of the chordæ tendinæ and valves, which are unequal in size.   He thus denies to the impulse any share whatever in the causation of the first sound.   In this opinion also I disagree with him.

Hyde Salter[*] (Fig. XV.) adopts the natural division of the cycle into ventricular systole, and ventricular diastole.   To the former he assigns one-third, and to the latter two-thirds of the whole.   The first sound is represented as of equal duration with ventricular systole, which is an obvious error, as a pause or period of silence, which I designate the "systolic pause," immediately succeeds the first sound, intervening between it and the second sound, and therefore of systolic rhythm.   The second sound occurs within the period of diastole, one-fourth of which,

* The death of this eminent man has just been announced (September, 1871), and I discuss his theory under a sense of depression at the great loss medicine has thereby sustained.

and one-sixth of the entire cycle, it represents. The pause, which coincides with ventricular diastole *minus* the second sound, and corresponds to one-half the cycle, immediately succeeds the second sound, and includes towards its termination the auricular systole, which therefore is diastolic in rhythm. This latter equals one-third of the pause, one-fourth of the diastole, and one-sixth of the cycle.

This scheme is in most respects faithful to nature, and correct in detail. I have already indicated one ground of exception to it, and in the scheme which follows others will be implied, which a comparison of the two figures (XV. and XVI.) will make sufficiently clear.

The plan which, after long and matured study, I have adopted, as most accurately representative of the natural grouping and

Fig. XVI.

a Ventricular systole   *   ...   .   *   = ⅓ of cycle.
b Ventricular diastole   *   ...   .   *   = ⅔ of cycle.
c Impulse and first sound   ..   .. = ¼ ventricular systole.
d Systolic pause   ...   . — ⅓ ventricular systole.
e Second sound   ...   .   incalculable.
f Diastolic pause   <··························> — ⅔ cycle less 2nd sound.
g Momentum of auricular systole ...   .   incalculable.

succession of phenomena, constituting a cycle of cardiac move-
ment, is set forth in the engraving (Fig. XVI.) As therein
exhibited, the cycle is divided into two primary parts, viz.:
ventricular systole, occupying one-third; and ventricular dia-
stole, occupying two-thirds of the cycle. Of the former, the
first two-thirds are covered by the impulse and the first sound,
and the last third by the short or systolic pause. Of the period
of ventricular diastole, the initial and the terminal portions are
occupied, respectively, by the second sound, and the momentum
or most energetic portion of auricular systole, both of which are
incalculable as to duration. Inasmuch as, in the normal state,
the contraction of the auricles is not manifested by any sensible
phenomenon, it is conveniently included within the period of
the long or diastolic pause, which commences at the end of the
second sound, and is characterized by the absence of all tactile
and acoustic signs.

Within the period of the first two-thirds of ventricular sys-
tole, coinciding with the impulse and the first sound, the arterial
valves are thrown open; the auriculo-ventricular valves are shut
down; the aorta and the pulmonary artery are distended by
efflux from the ventricles; and the auricles, partly by arrest at
the auriculo-ventricular openings, and partly by influx from the
cavæ and pulmonary veins, are likewise engorged. The circula-
tion in the coronary arteries is retarded; and that of the coro-
nary veins is accelerated. The pulsation of the great arteries of
the neck is virtually coincident with the impulse and the first
sound, although necessarily, by a brief but inappreciable interval,
posterior to them in time.

During the short pause which immediately succeeds the first
sound, and precedes the second sound, and which, according to
Marey, equals in duration one-tenth of a second, the contraction
of the ventricles and the distension of the auricles and arteries
continue; the arterial valves are still open, and those of the auri-
culo-ventricular orifices still firmly closed; temporary stasis
of the circulation in the cavæ and their immediate tributaries,
and in the pulmonary veins, takes place; the circulation in the
coronary arteries is still retarded, whilst that in the coronary
veins is quickened, and the radial pulse is perceptible. In

slow action of the heart the short pause may be readily recognized; in rapid action, and even at the ordinary rate, it is not so easily appreciated, and hence it has been, by many excellent observers, entirely ignored. Murmurs are occasionally, and not unfrequently, heard, which are most appropriately designated " post-systolic" and " pre-diastolic," as occupying, the former the initial, and the latter the terminal portion of this pause or period of silence. To this subject I shall have occasion to return. Dr. Sibson regards the short pause as coinciding with the commencement of ventricular diastole, and the reaction of the arteries; he says[*] : " A little consideration will make it evident that the sigmoid valves must come together at the very end of the systole, otherwise blood would at once flow back into the ventricle, during a period that, as M. Marey has shown in his important work on the circulation of the blood, precedes the second sound by one-tenth of a second, and coincides with the short pause. During this short pause between the first and second sounds, the walls of the arteries, just charged to the full by the systole, return upon themselves and drive the blood forwards into the smaller vessels, and backwards upon the arch and aortic valves." I believe, as already stated, that the ventricles, during the short pause, are still in a state of contraction, and the aorta and pulmonary artery in a state of distension by the ebbing current of blood; hence reflux cannot take place, though the valves be still open. MM. Chaveau and Favre conclude from their experiments, that ventricular diastole commences *with* the second sound, whereas the short pause is antecedent to it.[†] Coincident with the second sound is reaction of the aorta and pulmonary artery, closure of the arterial, and opening of the auriculo-ventricular valves, relaxation of the ventricles, and passage of blood from the auricles into the ventricles. The period occupied by the second sound is incalculably short. The long or diastolic pause succeeds the second sound, and with it covers two-thirds of the cycle. During this pause the arterial valves are still closed, and the auriculo-ventricular valves open; the ventricles

* The Movements, Structure, and Sounds of the Heart, 1869, p. 31.

† Traité de Physiologie, par F. A. Longet, 3ème Edition, tome, 2ème, 1869, p. 156.

are relaxed and in process of dilatation; undulatory contraction of the auricles, from the appendices towards the auriculo-ventricular openings, sets in and is continued, with passage of blood into the ventricles; and at its conclusion the auricles perform a brief but energetic act of contraction, the *momentum* of auricular systole, by which the ventricles are filled to distension, and reaction of their walls, constituting the succeeding ventricular systole, is induced.

I may here briefly state the result of my observations on M. Groux, who exhibited in his own person the pulsations of the heart, through a congenital fissure of the sternum, in Dublin, in the year 1858. On the 29th of February in that year I made the following notes. There was a vertical sulcus in the situation of the sternum, extending nearly its entire length, and bounded on each side by a continuous ridge of bone representing the margins of the sternum. The ensiform cartilage, or a flexible substance representing it, occupied the lower extremity of the fissure, which, at its widest part about midway between the upper and lower extremity, was an inch and a-half in transverse diameter; it was, however, susceptible of expansion to three inches by retraction of the shoulders, and by forced inspiration. Within this fissure a pulsating tumor existed, which was irregularly ovoid in figure, one and a half inch in its long, and half an inch in its short diameter, and did not extend laterally beneath the boundaries of the fissure. The long axis of this tumor extended from below and the left side upwards and to the right. The upper portion of the fissure was occupied by another and smaller pulsating tumor, seemingly continuous with the former. The superior extremity of the former of these tumors was on a level with the upper edge of the third costal cartilage, and its lower extremity with the middle line of the third intercostal space. During the period of enlargement this tumor communicated to the hand a feeling of increased hardness, and a rolling motion from above and right side downwards and to left; its pulsation was synchronous with the first sound, and followed by the second sound. The superior of these tumors pulsated first, and in an arc from above downwards and to the right, and the lower pulsation preceded the carotid and radial pulse.

Judging from the extent and course of the pulsation-wave, and from what may be observed in the dog, I regarded the inferior pulsating tumor as belonging to the right ventricle. The right auricle touches the anterior wall only by its appendix, and with a sudden "tap," during its diastole; it was soft at the moment of greatest distention; whereas, the line in which the remainder of the auricle presents itself to the anterior wall of the chest is from above downwards and to the right side. The superior tumor was the right auricle, which contracted first, in order of time, after the long pause, conducting a wave of blood into the right ventricle, the contraction of which immediately succeeded that of the auricle.

Dr. Pavy* holds that an ascending wave travels over the auricle immediately after, and communicated by, the systole of the ventricle; that this is promptly succeeded by a descending auricular wave running into the ventricle; that then the pause of ventricular diastole occurs, and next ventricular systole.

Dr. Pavy is, therefore, of opinion that in mammals, including man, auricular systole immediately *succeeds* instead of immediately *preceding* the systole of the ventricles, and that in this class no other contractile movement of the auricles occurs, although such is not the case in the frog and the tortoise.

I take the liberty of declaring that herein Dr. Pavy, excellent observer though he be, has fallen into error. An ascending wave of vibration, communicated to the blood of the auricle by the shock of ventricular systole through the tense auriculo-ventricular valves, is, no doubt, exhibited; it is equally true that a descending wave of contraction immediately succeeds the former, and conducts the first gush of blood into the ventricle after the retraction or opening of the auriculo-ventricular valve, or rather by the pressure of which mainly it is thrown open. The long pause now ensues; during its continuance blood still flows passively into the auricle and ventricle respectively; but now, towards the close of the long pause, auricular contraction of a more abrupt and vigorous character takes place, filling the ventricle to distention, and provoking an act of ventricular systole in which it is itself merged, and so on. This *presystole* or auri-

* *Medical Times and Gazette*, 21st November, 1857.

cular systole immediately preceding ventricular systole, is of the utmost moment diagnostically, as will appear in the sequel. I shall place in parallel columns, for the purpose of comparison, the succession of events as represented by Dr. Pavy and by myself.

PAVY.	HAYDEN.
Ventricular systole.	Ventricular systole
Ascending auricular wave (vibratory).	Ascending auricular wave (vibratory).
Descending auricular wave (contractile).	Descending auricular wave (contractile).
Pause of ventricular diastole.	Pause of ventricular diastole (long pause).
	Auricular systole (momentum of).

Between Dr. Pavy's views and mine, then, the difference consists in the denial by him of a continuous descending wave of auricular contraction, during the entire period of ventricular diastole; and, what is still more important, his repudiation of presystolic auricular contraction, both of which events I strongly insist upon.

The causes to which the two sounds of the heart have been respectively attributed by different authors, will be found briefly set forth in the subjoined table taken from *Longet's Physiology,*[*] and borrowed by that author from Barth and Roger. This table I have supplemented by the addition of the views of Dr. C. J. B. Williams, Dr. Bellingham, M. Chauveau, and my own.

	First sound.	*Second sound.*
Laennec, ...	Contraction of ventricles.	Contraction of auricles.
Turner, ...	Contraction of ventricles.	Shock of the heart rebounding against the pericardium in diastole.
Corrigan, ...	Shock of the blood against the walls of the ventricles in diastole.	Collision of the internal surface of the walls of the ventricles in systole.
D'Espine, ..	Contraction of the ventricles.	Dilatation of the ventricles.
Pigeaux (1832),	Shock of blood against walls of ventricles at the moment of diastole.	Shock of blood against the walls of the aorta and pulmonary artery at the moment of systole.
Pigeaux (1839),	Friction of the blood against the walls of the ventricles, the orifices and walls of the great vessels in systole.	Friction of the blood against the walls of the auricles, the auriculo-ventricular orifices, and the cavity of the ventricles, in diastole.
Hope (1834),	Mutual collision of the corpuscles of the blood in systole.	Mutual collision of the corpuscles of the blood, and impulse of blood against the walls of the ventricles, in diastole.

* Vol. ii., p. 153.

	First sound.	*Second sound.*
Hope (1839),	The sound of valvular tension, and of muscular extension, in systole.	The click of the sigmoid valves in diastole.
Rouannet, ...	The click of the auriculo-ventricular valves in systole.	The click of the sigmoid valves in diastole.
Piorry, ...	The entrance of blood into the left chambers of the heart.	The entrance of blood into the right chambers of the heart.
Piédagnel, ...	The contraction of the left ventricle.	The contraction of the right ventricle.
Carlile, ...	The sudden entrance of blood into the great arteries in systole.	The click of the sigmoid valves in diastole.
Magendie, ...	The shock of the apex of the heart against the side in systole.	The shock of the anterior surface of the heart in diastole.
Burdach, ..	Sudden entrance of blood into the ventricles containing air, in auricular systole.	The injection of blood into the arteries containing air, in ventricular systole.
Bouillaud, ...	The sudden tension and mutual collision of the auriculo-ventricular valves, and the sudden displacement of the sigmoid valves, in systole	The sudden tension and shock of mutual collision of the sigmoid valves, and the sudden raising of the auriculo-ventricular valves, in diastole.
Gendrin, ...	Vibration resulting from the collision of the blood in systole.	Percussion of the blood against the walls of the ventricles in diastole.
Cruveilhier, ...	The sudden raising of the sigmoid valves in systole.	The lowering of the sigmoid valves in diastole.
Skoda, ...	The shock of the blood against the auriculo-ventricular valves, and the impulse of the apex against the side. The shock of the blood against the walls of the aorta and pulmonary artery, in systole.	The shock of the column of blood against the walls of the ventricles, and the retrograde shock of the column of blood against the sigmoid valves, in diastole.
Beau, ...	The shock of the wave of blood against the walls of the ventricles, in ventricular diastole.	The shock of the column of blood arriving from the veins against the walls of the auricles.
G. Williams,	Muscular contraction of the ventricles in systole.	The shock of the returning column of blood against the sigmoid valves, in diastole.
Dublin Committee, 1835,	Friction of blood against the walls of the ventricles, and muscular contraction, in systole.	Tension of the sigmoid valves, and shock of the returning columns of blood, in diastole.
London Committee, 1836,	Sudden muscular tension of the ventricles, and shock against the chest, in systole.	Sudden closure of the sigmoid valves by the columns of blood in the arteries (in diastole).
Philadelphia Committee,	Muscular contraction of the ventricles, and click of the auriculo-ventricular valves in systole.	Closure of the sigmoid valves by the shock of the returning columns of blood in the aorta and pulmonary artery.

	First sound.	Second sound.
C. J. B. Williams,	Muscular contraction of ventricles, closure of auriculo-ventricular valves, and occasionally impulse of heart.	Click of sigmoid valves.
Bellingham,	Rush of blood through arterial orifices.	Rush of blood through auriculo-ventricular orifices.
Chauveau, ...	Sudden tension of auriculo-ventricular valves.	Sudden tension of sigmoid valves.
Hayden, ...	Shock of apex against wall of chest, and sudden tension of auriculo-ventricular valves and chordæ tendineæ.	Sudden tension of sigmoid valves.

M. A. Dumontpallier gives the following summary of the experiments of Chauveau, performed at Alfort on the 5th of September, 1860, in his presence, and repeated on the 29th of the same month in the presence, amongst others, of Doctors Corrigan and Banks of Dublin, with a view to determine the nature and cause of the sounds of the heart.*

A horse having been pithed, and artificial respiration maintained by means of a pair of bellows connected with the trachea, a "window" was made in the left side of the chest, and the pericardium laid open through this, so as to expose to view the left ventricle, left auricle, and the pulmonary artery. The auricle was seen to contract very feebly; but the ventricle with much energy, contracting from apex to base, and becoming globular, hard, and corrugated on the surface. The left wall of the ventricle was forcibly elevated in such a manner that the organ must have struck the wall of the chest. A stethoscope applied over the ventricle enabled the observer to hear two sounds; the first dull and prolonged, and coinciding with ventricular systole; the second sharper and shorter, coinciding with ventricular diastole.

After the first sound "a short silence" ensued, and after the second, "a longer silence." When the root of the pulmonary artery was taken gently between the finger and thumb at the moment of the second sound, a vibration was felt, and a stethoscope applied here at the same moment enabled the observer to hear the second sound. If, at the moment of systole of the ventricle and occurrence of the first sound, the roof of the left auricle

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* *Gazette des Hospitaux; Dublin Hospital Gazette*, 1st November, 1860.

were pushed down with the finger, the vibration of the auriculo-ventricular valve was felt. If now an incision were made in the auricular appendix, and the finger introduced through it into the auriculo-ventricular orifice, it was felt to be grasped at the moment of systole by the free edges of the valves, and held for a period corresponding in duration to the first sound, which was either suppressed or weakened, owing to suspension or impairment of the normal vibration of the valves. The reporter adds: " From this experiment may we not conclude that the first sound is owing, almost entirely, to the contraction of the auriculo-ventricular valves ?" A query which, I apprehend, few if any will answer in the negative.

Chauveau next proved, by mutilation of the valves, that abnormal sounds depend upon disease of these. Thus, when closure of the mitral valves was prevented during ventricular systole, by means of a small steel rod, blood rushed back into the auricles, and a *bruit de soufflet* was the result. Tearing of the valves, or dividing the chordæ tendineæ was followed by a similar result. Systolic murmur likewise followed compression of the pulmonary artery during ventricular systole.

In a similar manner he caused diastolic murmur by keeping the sigmoid valves apart by means of a fine instrument, by pinning them up to the wall of the artery, or by destroying them. In every such instance blood rushed back into the ventricle and produced diastolic murmur.*

Marey and Chauveau† have determined, by experiments on the horse, the relative duration and precise relationship of the several movements of the heart.

The systole of the auricles precedes that of the ventricles by two-tenths of a second. Ventricular systole and impulse commence together, but the former exceeds the latter in duration by one-tenth of a second, the length of ventricular systole being three-tenths of a second.

They have also proved experimentally that the force of contraction of the right ventricle is to that of the left ventricle, in the horse, as 1 to 3. The relative proportion of the various car-

* See *Gazette Médicale*, 1856, for Chauveau's memoir on this subject, *in extenso*.

† *Longet's Physiology*. 1869, vol. ii. p. 146.

diac movements is, according to these eminent authorities, the same in man as in the horse.

It will be seen by reference to Fig. XVI. that the conclusions at which I had arrived independently, from observation of the healthy human heart, are nearly identical with those just detailed.   I did not see the report of Marey and Chauveau's experiments till very recently (1868), and I am therefore entitled to regard them as eminently confirmatory of my own views.

CHAPTER II.

PHENOMENA OF THE HEART'S ACTION IN RELATION TO THEIR MODIFICATION BY DISEASE.

I SHALL now briefly discuss, individually, the cyclical series of phenomena which constitute a single action of the heart.

The impulse of the heart, which, in the state of health, may be perceived by the application of the hand in the fifth intercostal space of the left side, about two inches below the level of the nipple, and one inch inside the nipple-line, is characterized by a gentle lifting motion over an irregularly circular space of about one inch in diameter. This is due to the impact of the apex and adjacent portion of the anterior surface of the left ventricle, against the chest wall; commences with the systole of the ventricles, which exceeds it in duration by one-third; and strictly corresponds in time with the first sound. As the systole is equal to one-third of the cycle, so the impulse equals one-third of the diastole.

There are at least three positive varieties of abnormal impulse, besides those of a negative character, namely: the prolonged, diffused, heaving, and *double* impulse of left ventricular *dilated* hypertrophy; the abrupt but prolonged and heaving impulse of left ventricular *simple* hypertrophy; and the abrupt hammering impulse of nervous palpitation.

The impulse is liable to impairment, suppression, and displacement. It is obscured or enfeebled in fatty degeneration of the heart, and in atrophic attenuation of the left ventricular walls. In the advanced stages of the former of these conditions it may be entirely suppressed, and likewise in the worst form of typhus fever characterized by suspension of the first sound.

Displacement of the apex of the heart, to whatever cause due, will involve change of the seat of impulse, and may take place in the direction upwards, downwards, towards the right, or towards the left side.

Displacement of the heart to either side is most frequently the consequence of liquid or gaseous effusion into the opposite pleura. Two different opinions are held as to the mode in which this displacement is effected. One, and that which is almost universally accepted, is that the heart is displaced by mechanical *detrusion* to the opposite side; and the other, to the effect that it is *drawn* towards the sound side by the elastic tension of the unaffected lung.

This latter view has been ably advocated by Dr. Douglas Powell,* who considers the median position of the mediastinum, in the state of health, to be maintained by a well-balanced tractile force exercised upon its opposite sides by the elastic tension of the lungs. When either lung, therefore, passes into a state of collapse, whether from liquid effusion or aeriform accumulation, the normal balance of pulmonary tension is of necessity deranged, and the mediastinum and heart are *drawn* towards the unaffected side by the unopposed tension of the sound lung. In pneumothorax the displacement is instantaneous; but he admits that " beyond this it may be increased by the direct pressure or subsequent accumulation of air." In support of this doctrine the following experiments are adduced:

Experiment 1. In a fresh subject, a needle was thrust through the pericardium at the left margin of the sternum; an opening was then made in the left pleura, through which air was sucked in, and immediately the needle was deflected to the left. The arms were now drawn up equally, and an act of inspiration thus artificially made after the manner of Sylvester, and the deflection of the needle to the left was observed to be immediately increased.

Experiment 2. A dog was brought under the influence of chloroform, a needle was inserted into the pericardium, and the pleura laid open as in the former experiment, and with a similar result, namely, deflection of the needle towards the injured side, to the extent of 20°.

It seems to me that in Experiment No. 1 the deflection of the needle to the left was directly and exclusively due to the pres-

* *Medical Times and Gazette,* 30th January ; 13th and 20th February; and *British Medical Journal,* 17th July, 1869.

sure upon the mediastinum of the air just admitted into the pleura; and the increased deflection exhibited on drawing the arms upwards was the result of increased pressure by the introduction into the pleura of a further volume of air.   This air was free, and its pressure therefore not neutralized by the elastic reaction of the lung, as was the case with that included within the pulmonary tissue of the opposite lung.   The right lung was, moreover, in the first experiment, in the state of maximum expiratory contraction characteristic of the dead body, and its elastic tension proportionately reduced.   In this condition, the elastic reaction of the lungs of the dog was found by Hyde Salter equal to a pressure of only four inches of water.*   The elastic tension of the lung is an expiratory force, increasing with its expansion, and tending towards a common centre.   No doubt this centre is in the root of the lung, but external to the mediastinum.   Here, therefore, the elastic reaction of the lung has its point of rest, and beyond this in the direction inwards the range of its elastic tension does not extend.   The length and extensibility of the blood vessels and air-tubes of the lungs, and their capacity for physical reaction, are strictly adapted in the normal state to the dimensions of the pleural cavity and the position of the mediastinum.

In reference to Experiment No. 2, which is in no degree less open to all the objections above-stated, I have in addition to remark, that no mention is made of oscillation of the needle from right to left alternately with inspiration and expiration, which must have taken place if deflection depended upon the movements of the right lung.   As to the alleged effect of sudden introduction of air into either pleura, in causing "partial collapse of the opposite lung, by removing resistance to its elastic tension," I cannot perceive how the position of the mediastinum can be in any degree thereby influenced, save in so far as the pressure of free air on the diseased side must determine its displacement towards the opposite.   Surely, even though partial collapse *per se* could in any degree influence the position of the mediastinum, complete collapse on the opposite side would be more-than competent to neutralize it.

* *Lancet*, 5th August, 1865.

Dr. Powell published two cases * of pyo-pneumothorax, illustrative of displacement of the mediastinum by "elastic recoil" of the opposite lung. By means of a water-pressure guage he showed that no air-pressure was exercised upon the mediastinum on the diseased side of the chest. But pus was found on that side in both cases, and in one of these to the amount of a pint.

With a view to satisfying my mind as to the validity of the objections above urged against Dr. Powell's doctrine, and as to the degree, if any, in which inspiratory expansion of either lung, the other being functionally in abeyance, was capable of influencing the position of the mediastinum, I performed, on the 14th October, 1871, the following experiment, with the assistance of Mr. Ward, one of our demonstrators of anatomy, and in presence of the anatomical class in the School of Medicine, Cecilia-street.

The anterior wall of the chest of a subject brought in for dissection was removed, with the exception of the sternum, which was divided with the saw by a median incision from the upper to the lower extremity. The edges of this incision were divaricated by means of a wedge, to the extent of an inch, and a piece of copper wire, nine inches long, made sharp at one extremity, and rolled into a coil at the opposite, was inserted perpendicularly into the pericardium, and fixed in the substance of the heart. The left lung was now detached from its root, and the mouth of the left bronchus plugged with a cork, which was held in position and made air-tight by means of a ligature passed round it. Through the outer extremity of the cork a string was passed, by means of which it might be instantaneously removed. Through an incision made in the trachea at the root of the neck a large glass tube was introduced, and the right lung slowly inflated, the subject lying upon its back. The position of the wire fixed in the pericardium was not in the smallest degree influenced till towards the point of maximum inflation, when the anterior edge of the lung advanced and began to press against the side of the pericardium. The free extremity of the wire now began to deviate to the right, and at the end of inflation had moved fully an inch towards the right side; and on allowing

* *Medical Times and Gazette*, 21st August, 1869.

the lung to collapse by its elastic reaction, through escape of the air from the opening in the trachea, the needle gradually returned to its original position. The process was repeated several times, with the same result. At the moment of maximum inflation the cork was now suddenly withdrawn from the bronchus, and the lung thus permitted to collapse abruptly, as if by rupture of the opposite lung; but the result was in no degree altered, save in regard to the rapidity of return movement of the lever from right to left.

The obvious conclusions to be drawn from this experiment are the four following: First, that in the first stage of inspiratory expansion of the lung, the position of the mediastinum is in no degree affected; second, that towards the end of the second and final stage of expansion of the lung, the mediastinum is displaced towards the *opposite* side by pressure of the anterior edge and internal surface of the inflated lung; third, that at no stage of inspiration is the mediastinum displaced by *traction towards* the expanding lung; and, fourth, that the sudden exhaustion of one lung, even in the stage of maximum expansion by rupture of the opposite lung, is incapable of determining by recoil or otherwise, a movement of the mediastinum towards that side, beyond the point of equilibrium.

Having now shown, as I conceive, good reason for adhering to the doctrine of lateral displacement of the heart by pressure, as promulgated by Bertin, Laennec, Hope, Stokes, Walshe, and indeed by the great majority of writers, and as almost universally held at the present time, I proceed, after the example of Stokes,[*] to classify all cardiac displacements whatsoever under the two heads of "excentric" and "concentric."

The former are caused by diseases "of accumulation," solid, liquid, or gaseous, external to the pericardium, and pressing *ab extra* upon the heart.

The latter are due to contraction in volume of either lung, and a concentric movement of adjacent parts, including the heart, to fill the vacuum thence potentially arising.

The excentric displacements, as the name implies, are therefore dependent upon a force operating in a direction from its

* *Opus citat.*, p. 458.

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seat; whilst those named concentric are no less obviously due
to a cause acting by traction towards the same point. Excen-
tric displacements may take place in the four opposite directions,
upwards, downwards, to the right, or to the left side; and dia-
gonally or intermediately to these lines. Upward displacement,
direct or oblique, of the heart by pressure, is most frequently
caused by enlargement of the left lobe of the liver, hydatid
tumor of that organ, perihepatic abscess, aneurism of the in-
ferior thoracic aorta,* and temporary paralysis of the left ala of
the diaphragm, with ascent of an inflated stomach in diaphrag-
matic pleuritis.†

Bouillaud declares that upward displacement of the heart may
be caused by ascites in an extreme degree.‡ I have never wit-
nessed notable elevation of the heart from this cause. Displace-
ment downwards is, when present, usually the result of pulmonary
emphysema engaging pretty equally the anterior portion of both
lungs; by the diagonal pressure so exercised, as well as by the
general depression or flattening of the diaphragm, the heart is
displaced downwards, and occupies a position behind the xiphoid
cartilage. Downward displacement may likewise result from the
pressure of an aneurismal or other tumor occupying the upper
portion of the anterior mediastinum.

Excentric displacement to either side is most frequently due
to liquid accumulation in the opposite pleura. In the effusion
consecutive to acute inflammation, Dr. Stokes has shown, that
the relaxation of the mediastinum involved in the acute inflam-
matory process may determine lateral displacement of the heart
at an early period of the disease, and under the comparatively
slight pressure of an undistended pleura. In all such cases the
heart is dislocated in the horizontal line, and occupies a position
under the right nipple when the effusion is on the left side; and
when on the right, the heart may be felt pulsating at a greater
or less distance outside the left nipple-line. Doctor Douglas
Powell has shown§ that in the right lateral displacements the

* See case (Fay), chapter ix.
† For a case in illustration, see an article by the author, entitled "Diaphragma-
tic Pleuritis," *Dublin Medical Journal*, August, 1871.
‡ *Traité des Maladies du Cœur*, tom. ii., p. 531.
§ *British Medical Journal*, July 17, 1869.

apex of the heart is depressed, but never so as to occupy a position external to the base; whereas, in displacements to the left side, the apex is relatively elevated, and the long axis of the heart nearly or quite horizontal.

Cases of this kind are characterized by percussion dullness on the side whence the heart has been displaced, or posterior in the line of transit of the organ, and commensurate with the extent of its displacement; by clearness on the opposite side in advance of the organ; and where the liquid is absorbed or otherwise removed, by return of the heart to its natural position with corresponding rapidity.

The functions of the heart and the attendant phenomena are in no degree modified by its abnormal position, with the following exceptions: when placed in front of, and in contact with a solid tumor, such as an aneurism, a solidified lung;[*] or the spinal column,[†] its impulse may be so vigorous and diffused as to lead to the diagnosis of hypertrophy. Hope has also noted, in a case of dexiocardia, the existence of systolic murmur, which ceased on the return of the heart to its normal position; and was due, as he believed, to twisting of the aorta in its altered relationship to the heart.[‡]

Consecutively to the removal of the displacing fluid by absorption or otherwise, the heart not unfrequently has been known to transgress its normal boundary, and pass considerably to the opposite side. This occurs where the lung has been compressed, and rendered incapable of expansion, after removal of the compressing medium, by previous obliteration of its vesicular structure; or by the constricting agency of dense false membrane. Such a phenomenon is, *mutatis mutandis*, analogous to the retraction of the features from consecutive atrophy and shortening of the paralysed muscles, in unilateral paralysis of the face. The distinctive features, then, of lateral displacement of the heart by liquid accumulation in the opposite pleura, are, the slow and mensurable mode of its occurrence; dullness on the side whence it has been displaced, and clearness on the opposite side beyond the limits of cardiac dullness; and in the event of removal of the displacing medium, return of the heart to its natural situation;

[*] Stokes.　　　　[†] Hope.　　　　[‡] *Opus citat.*, p. 536.

or beyond it where the lung, previously compressed, has become incapable of expansion.

Lateral displacement of the heart, by detrusion, may be likewise caused by cancerous growths in connection with either lung or pleura. In Dr. Houston's case a cancerous tumour of the right lung was the sole cause of displacement of the heart to the left side.*

In most cases, however, of displacement of the heart by pressure connected with cancerous growths of the lung or pleura, but especially the latter, serum was likewise present in large quantity, and presumably was the principal agent in displacement. Case 20 (Rourke), affords a notable example of this kind. Where, however, the tumour is of the nature of soft or medullary cancer, which, by its expansion and elasticity is capable of exercising diastolic pressure, displacement of the heart may be, and usually is, effected without the intervention of liquid accumulation. Cancerous disease of the lung or pleura, operating as a cause of displacement of the heart, and associated or not with serous effusion, is characterized by certain symptoms of great diagnostic value; these are especially the two following, namely, engorgement of the subcutaneous veins of the affected side of the chest; and distinct transmission, over the same side, of the sounds of the heart. Associated with dullness on percussion; absence of respiratory sound save at the root of the lung, and of vocal fremitus; and with excentric displacement of the heart; the signs above indicated become pathognomonic of cancer of the lung or pleura.

Thoracic aneurism may be likewise a cause of displacement of the heart to either side, but most frequently the right, because when capable of making lateral pressure upon the heart, its usual situation is the posterior mediastinum and left pleural cavity. The diagonosis of this cause of displacement of the heart cannot be difficult.

The heart may be likewise displaced to either side by the pressure of free air in the opposite pleural cavity, constituting pneumothorax; by pulmonary emphysema preponderating on one side, and engaging the anterior and inner portion of the lung; and by passage of the hollow abdominal viscera into the thorax.

* See *Catalogue of Museum of Royal College of Surgeons, Ireland.*

Dr. Stokes is of opinion that simple pneumothorax, without fistula in the lung, can cause displacement of the heart only when the aeriform collection is large in quantity ; and that, with fistula, it can produce this result only where the egress of air is prevented during expiration, that is, where the fistula is valvular.

I incline to regard this view as somewhat too exclusive. Free air in the cavity of the pleura, with or without fistula of the lung, and at the temperature of the living body, must undergo considerable expansion during inspiration, and a high degree of compression during expiration In the former condition the mediastinum, as the most yielding wall of the hemithorax, is liable to undergo displacement under the pressure of elastic expansion of the air, no longer neutralized by the resiliency of the lung and pulmonary pleura ; and in the latter state, or that of expiration, under the compressing force of the reacting thoracic walls the contained air must undergo a high degree of condensation, and the mediastinum must sustain an equivalent pressure on the corresponding side.   These forces, in succession, would operate continuously.   A fistular opening in the lung of the average diameter, even though direct and patulous, is by much too narrow to neutralise, though it may modify this pressure, by evacuating the pleura within the period of an ordinary expiration.   In order to effect this, it must bear the same proportion to the pleural cavity which the trachea does to the expanded lung ; and even in that case a certain degree of obstruction to the exit of air would be necessarily presented at the fistula, and an equivalent centrifugal pressure sustained by the parieties of the pleural cavity, owing to the absence of the gradual and well-adapted transition of cubic space exhibited by the bronchial system in its relationship to the trachea.

Simple pneumothorax, then, is of itself, and *a fortiori* in conjunction with liquid, in my opinion competent to effect lateral displacement of the heart.   After death, owing to the cooling and condensation of the air within the pleura, the heart will have returned in some degree towards its natural situation. In such a case, therefore, the position of the heart, as exhibited by *post mortem* examination, will not accurately represent its situation at the time of death.   At the latter period the dis-

placement is greater, owing to the alternate expansion and condensation of the æriform contents of the pleura, under the combined influence of heat and compression. Emphysema of the lungs, if greatly in excess on one side, and engaging the anterior portion of the organ, may become the agent in lateral dislocation of the heart; but this affection is usually bilateral, and pretty equally balanced on both sides of the chest; hence the cardiac displacement resulting therefrom consists usually in depression of the organ as previously mentioned. Increase in the volume of the lung, and consequent displacement of the heart, may be excluded from the category of results of inflammation of the substance of the organ. Nevertheless, an example has been adduced by Professor R. Smith[*] of such increase in the volume of the right lung in "plastic pneumonia" that the liver and diaphragm were considerably depressed into the abdomen. In such a case the heart could hardly escape displacement. Diaphragmatic hernia, involving intrusion of the abdominal viscera into the chest, completes the category of causes of lateral displacement of the heart. Comparatively few examples of this singular malposition are on record, and these have been all associated with partial deficiency of the diaphragm.

Bouillaud quotes from Weyland the following illustrative cases:

"In an infant which died some hours after birth, cadaveric examination revealed that the stomach, a great part of the intestines, the spleen, and the pancreas, were contained in the left side of the chest. The right side included the heart, the thymus, and the two lungs."

"An infant of seven years died of vomiting, which had continued from its birth, and ultimately became complicated with cerebral disease. On opening the body it was found that the cavity of the left side of the chest, to the level of the second rib, was filled with the convolutions of the intestines. The heart was situated in the middle line. The left lung, atrophied, presented scarcely a sixth part of its normal volume. The right lung was well formed, and presented nothing unusual."[†]

The remarkable case of phrenic hernia with displacement of

[*] *Dublin Journal of Medical Science,* vol. xix.
[†] *Traité Clinique des Maladies du Cœur,* 1835, tom. ii. p. 524.

the heart to the right side, from congenital imperfection of the diaphragm in a female aged 75, recorded by Cruveilhier,* deserves special notice, as being in many respects the most singular example of the kind to be found in medical literature.

The following remarkable example of congenital malposition of the abdominal and thoracic viscera occurred in the practice of Dr. Osborne, and has been quoted by Drs. Stokes and Graves.† I give it in the words of Dr. Stokes:

" A man about 40 years of age died of tubercular phthisis (in Sir Patrick Dunn's Hospital). The œsophagus, after passing through the usual opening in the diaphragm, was found to re-enter the thorax by another very large opening in the tendinous expansion towards the left side. The stomach, of which the cardiac and pyloric extremities were approximated, occupied the inferior portion of the left thoracic cavity.

" A considerable portion of the transverse arch of the colon was also included in the left side of the chest. These viscera, loosely but permanently fixed by means of the serous membranes, all rested on the convex surface of the diaphragm, and had pushed the heart and mediastinum towards the right side.

" The margin of the unnatural opening in the diaphragm was formed by a round tendinous cord, about the thickness of a quill, which added greatly to its strength, and was evidently of very ancient formation. The lung, small and tuberculated, did not exhibit any signs of compression, and was adherent to the abdominal viscera. It may be easily conceived that the left pleural cavity was continuous with the cavity of the peritoneum, and both were lined by the one serous membrane."

Dr. Stokes remarks, " This case is pregnant with interest; we observe in the adult *a new cause of displacement of the heart*, and a new source of difficulty in stethoscopic examination; for it is quite evident that auscultation applied to the left side of the thorax would have furnished very fallacious information, and the sounds heard would have varied according as the stomach and colon were full or empty. The same observation applies also to percussion; and the fact is that during the life of this

* *Anatomie Pathologique*, livraison 17.
† *Dublin Hospital Reports*, vol. v.

patient, those who examined his chest could not reconcile the phenomena afforded by auscultation or percussion with any known disease of the chest. The respiration was heard everywhere, except inferiorly and anteriorly on the left side, and here percussion gave a clearer sound than natural. No râle was audible in this part of the chest, but borborygmi and sounds resembling those produced by the motion of fluids in the intestines were observed."[*] This is the case on which Graves and Stokes with good reason dwell as affording conclusive evidence of the competency of the stomach to accomplish the act of vomiting, unaided by the abdominal muscles, as vomiting had repeatedly occurred during the man's last illness. Dr. Stokes makes the important remark, " In the case of imperfectly developed diaphragm, in congenital dexiocardia, and again, in the transverse displacement from disease, the functions of the heart are not manifestly injured by the change of position."[†]

In the case just detailed, if a suspicion had been entertained as to the cause of thoracic tympany, the crucial test of causing the patient to swallow some liquid, whilst the ear or stethoscope was applied to the left chest, might have been used, and the metallic tinkle of liquid entering the stomach within a few seconds after the visible movement of pharyngeal contraction, would have removed all doubt.

Concentric displacement of the heart consists in a movement of the organ *towards* the side whence the displacing force operates. Exception may be taken to this definition on the ground that the displacing force, atmospheric pressure, acts from without, and from all points of the circumference of the chest. This objection would be strictly valid. Nevertheless, the above admission having been expressly made, the impugned definition, which is found convenient, may be retained without prejudice to the interests of truth.

Concentric displacement is usually effected, not horizontally, but diagonally upwards and to one side. This is, no doubt, due to the fact that adhesion of the contracted lung to the cone of the pleura, has, in the majority of such cases, taken place pre-

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[*] *The Diseases of the Heart and Aorta*, 1854, p. 456.
[†] *Opus citat.*, p. 458.

viously to its recession. The causes of concentric displacement
may be classified under two heads, namely,

1. Gradual decrease in the volume of the lung from intrinsic
   or substantive disease of the organ.
2. Compression of the lung by liquid or air in the pleura, and
   subsequent obliteration of its vesicular structure by simple
   condensation, or by constriction of false membrane; with
   removal of the compressing medium.

The causes which belong to the former category are these:

1. Tubercular atrophy of the lung, with or without cicatriza
   tion.
2. Cirrhosis of the lung.
3. Chronic gangrene of the lung.

Tubercular atrophy of the lung, as a cause of concentric dis-
placement of the heart, has been well illustrated by Dr. Stokes,
who explains the absence of cardiac displacement in ordinary
phthisis by the usual presence of the disease in both lungs, and
by the inflation of cavities in its advanced stages. The cases in
which it is most likely to be presented are those familiar to
every physician much conversant with thoracic disease, in which
a slow process of wasting of the lung substance, accompanied
with softening of small and distinct masses of tubercle, and for-
mation of minute disseminated cavities, occurs. In such cases
there is extreme flattening and retrocession of the anterior wall
of the chest on one side, whilst the physical signs are those of
small cavities dispersed throughout the lung.

Dr. Stokes dwells upon the fact that in many such cases cica-
trization of the cavities takes place, and, in either view, and
perhaps likewise in some measure from the comparative im-
munity of one lung, the patients not unfrequently survive many
years, in a state of broken health, which ordinarily ends in death
within as many months.

Many cases of this kind have come under my notice, and in
most of them I have observed a proclivity to copious hæmop-
tysis. The patients sometimes cease to cough and actually
recover flesh, especially on removal to the country; but the
physical signs remain unaltered.

The direction of displacement from the cause last mentioned

is diagonally upwards, where, as usually happens, adhesion of the apex of the lung to the cone of the pleura has previously taken place. Whether displacement of the heart be due to traction of the retreating lung, as is generally held to be the case, or to atmospheric pressure, as I believe it is, the diagonal movement would be equally explicable, because in either case the receding chest-wall and the adjacent viscera would follow the retracting lung.

Cirrhosis of the lung, or dilatation of the bronchial tubes with condensation and atrophy of the pulmonary substance, and recently named "fibroid phthisis," by Dr. Andrew Clark, is the most frequent cause of concentric displacement of the heart. It operates in the same manner as chronic tubercular atrophy; but with this difference, that displacement is usually effected in the horizontal line, and because, as I believe, antecedent pleuritic adhesion to the apex of the pleura is not common.* The diognosis between the two last-mentioned causes will be found in many cases difficult, and must rest mainly upon the evidence of actual excavation upon the one hand, and upon the special symptoms and signs of cirrhosis on the other; amongst which I rank in the first place, a state of general and varicose engorgement of the superficial veins of the extremities, associated with great emaciation. Displacement of the heart, consecutive to the removal of air or liquid, may be best diagnosed by reference to the previous history, if such can be obtained from an accurate and reliable source. The displacement is always towards the sound side of the chest, in the first instance, and subsequently towards the affected side; whereas concentric displacement is primarily towards the affected side, and is permanent.

Backward displacement of the heart may take place as the result of tumors in the anterior mediastinum;† collections of air, pus, or blood in the same situation, or a posterior exostosis of

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* Dr. Hughes exhibited before the Pathological Society of Dublin, in the Session 1873-4, an example of cirrhosis of the left lung, with displacement of the heart to a point beneath the left clavicle. But in this case there were several vomicæ in the lung, which was adherent to the cone of the pleura.

† See Case 64 (A. Murray) "Cancer in Anterior Mediastinum."

the sternum. Normally, the point of apex-pulsation varies slightly with the position of the body. Thus, in the supine posture the apex recedes somewhat, and its impulse is less distinctly felt; in the prone position, on the contrary, it is brought by the weight of the heart into persistent contact with the anterior wall of the thorax; and in the lateral positions it gravitates about half an inch to the corresponding side.

As to the cause of the stroke of the heart against the side, on which the phenomenon of impulse depends, the most plausible, because apparently the most scientific, theory, is that first propounded by Sir James Alderson, and now known as that of Gutbrod. This is the recoil theory, and is identical with that by which the recoil of firearms, on being discharged, is explained. In the latter case, the recoil is due to the sudden derangement of equality of pressure on the internal surface of the arm at the moment of its discharge, which results from the absence of an opposing wall at the point of exit. The consequence is, that whereas at all other points centrifugal pressure is neutralized by equal pressure upon opposite points, the pressure made upon the surface opposite to, and equal in extent with, the orifice of exit, is not so neutralized, and tends to propel the entire body in the direction in which it operates. Unfortunately, however, for the ingenious authors of this theory, the simplicity of which so strongly recommends it to our acceptance, the parallel cannot be sustained; the recoil of firearms is in fact, and perceptibly, posterior in time to the explosion, as it must be if dependent upon the agency above explained; whereas the impulse of the heart is strictly synchronous and coeval with the first sound. I do not wish to deny that on the principle stated, a recoil of the apex must and does take place; but it is in the direction of the orifice of the aorta, i.e., downwards, *back-wards*, and towards the left, and in time perceptibly later than the first sound; it is the force by which the ventricles are elongated after systole, and the apex lowered to the position which it occupied previously to that act. It is well exemplified in pericardial frottement at the apex, which is usually characterized by a double rub, the first element of which coincides with the impulse and the first sound; and the second with the descent

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of the apex, and is an abrupt, vigorous, and manifestly a "back" rub.

Dr. Sibson maintains that, during the impulse, the apex and the anterior walls of the ventricles move forwards by a lever movement, the fulcrum being the base of the pericardium attached to the central tendon of the diaphragm, and kept tense by attachment above to the investments of the great vessels. He says, the apex moves forwards and to the right, with a revolving movement upwards; there is a movement forwards and outwards, "the ventricles over their whole surface make a direct thrust against any object upon which they impinge, and wherever, therefore, they come in contact with the walls of the chest." The impulse is, therefore, in his opinion due to the impact, not alone of the apex, but of a great portion of the anterior surface of the ventricles. The cause of impulse he considers to be quintuple, viz.:

1. Muscular rigidity.
2. Lever movement of ventricles.
3. Outward pressure of blood.
4. Movement of repulsion (Alderson, Gutbrod, and Skoda).
5. Lengthening downwards of blood-column in aorta and pulmonary artery.*

Pettigrew has shown that the fibres of the ventricles commence and end at the ventricular orifices; they therefore form elongated loops, the extremities of which are fixed at the base, whilst the looped portion, forming the apex, is free. Now, the direction of the heart's axis in a state of quiescence being downwards, forwards, and to the left, at an angle of about 15°, and the anterior wall of the chest forming a nearly vertical plane in front of it, the contraction of a group of such fibres, interlaced at the apex, must, by raising the apex towards their fixed points at the base, bring it into forcible collision with the anterior thoracic wall, at a point somewhat above the level of their loops in a state of repose. Hence the impulse of the heart.

Double impulse, as already stated, is associated with left ventricular hypertrophy of the excentric form. It is due to strong diastolic reaction of the left ventricle at the moment of inci-

* *Medical Anatomy*, last fasciculus, 1869.

pient diastole, and coincides, therefore, with the period of ventricular recoil, according to Gutbrod's theory.

The ventricle being hypertrophied, and its cavity dilated, it is readily conceivable that, as the volume of blood entering it is in proportion, its reaction or recoil would involve a second impulse. Two forces are, no doubt, in operation here: the reaction of discharge as already explained, and the action of influx from the auricle at the moment of cessation of ventricular systole. Quite irrespectively of the explanation just offered, in regard to which difference of opinion is admissible, the phenomenon of double impulse is pathognomonic of dilated hypertrophy of the left ventricle, as yet in a state of tissue-soundness.*

The first sound of the heart coincides with the impulse and the first two-thirds of ventricular systole; it is the product of a two-fold cause; namely, the impulse of the ventricles, and the sudden tension of the auriculo-ventricular valves and chordæ tendineæ. Other causes of sound, at the moment of ventricular systole, have been insisted upon; for example, the mutual friction of the fibres of the ventricles, giving rise to the muscular *susurrus;* the rush of blood over the rough surface of the ventricles, and through the arterial orifices; and the sudden raising of the sigmoid valves.

The last-mentioned cause cannot, in any degree, contribute to the first sound, because the only sonorific element of valvular movement, namely, valve-tension, is not involved in it. The valves are raised by a pressure *ab intra,* but are in no degree subjected to tension; hence, they can contribute no factor of systolic sound, unless by obstruction, which implies disease.

The sound produced by the movement of blood over a rough surface is certainly not of the character of the first sound of the heart, which seems to be a compound of a "thud" and a

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* Dr. Stokes has called attention (*Diseases of the Heart and Aorta,* p. 328,) to an impulse of a peculiar character, which is occasionally met with in cases of extreme softening and attenuation of the left ventricle; the impulse is diffused, weak, and flapping, and suggests rather the passive shoc of an aneurism than the active impulse of the ventricles. This observation I have often verified; it is of great diagnostic value.

" click;" the former, as I believe, caused by the impulse of the ventricles, and the latter by the sudden tension of the auriculo-ventricular valves and chordæ tendineæ.

The theory of muscular susurrus, as applied to the heart in explanation of its first sound by several writers, and recently advocated with so much ability by Professor Haughton,* implies asynchronism of contraction of the fibres of the ventricles, as in voluntary muscles; otherwise, manifestly, intrinsic friction could not occur in the ventricular walls. But there is no asynchronism; the fibres of the ventricles contract simultaneously, and hence they develop no sound of interfascicular friction. The theory of mutual collision of the blood-corpuscles scarcely deserves notice; a sound produced by such a cause would be of a blowing character, as is the basic murmur of anæmia; but in health the first sound of the heart partakes in no degree of this character.

The duplex constitution of the first sound, and the twofold cause to which it is due, as above stated, viz., the impulse of the ventricles and the tension of the. auriculo-ventricular valves, are, to my mind, satisfactorily proven by its occasional reduplication, to which special reference will be made further on; suffice it here to say, that reduplication of the first sound is the result of its resolution into the above-mentioned two elements.

Dogiel and Ludwig conclude from some experiments performed by them, that the first sound is due to the contraction of the muscular fibres of the ventricles exclusively, and that valve-tension has no share in its production. Dogs were poisoned with curara, the heart was exposed, and artificial respiration was maintained. The venæ cavæ, pulmonary artery, pulmonary veins, and aorta, were successively ligatured at their junction with the heart. The heart was then removed from the body of the animal, and placed in a glass vessel filled with defibrinated blood. By means of a peculiar arrangement, a stethoscope was made use of for the purpose of auscultating the heart within the vessel. As long as the heart pulsated, a first sound, but faint, was heard. They declare that previous to

* *Outlines of a New Theory of Muscular Action*, 1863.

the removal of the heart from the body a similar result was obtained.*

In reference to the foregoing, Guttmann announces that he has repeated the experiments of Dogiel and Ludwig, with, to his mind, a contrary result. He declares that in the bloodless heart the systolic sound has a character quite different from that produced in the heart when the blood is flowing through it, and causing forcible closure and tension of the valves; but even in the bloodless heart, he maintains that valve-tension is not entirely excluded, inasmuch as when it contracts, the papillary muscles continue to contract and tighten the valves.†

There is much force in the foregoing remarks of Guttmann, but in other respects the experiments detailed are singularly inconclusive. Deligation of the vessels connected with the heart, even if this were done in the order of the circulation, could not have been accomplished within the period of a single cycle of cardiac movement, so as effectually and completely to exclude blood from the heart; and if this were not effected, valve-pressure and tension became inevitable, so long as the contractions of the heart continued. Hence, the valvular element of sound cannot be regarded as excluded in these experiments.

Reduplication of the first sound of the heart may be exhibited in four different connections:

1st and oftenest, it is associated with simple functional derangement of the heart, as evinced by nervous palpitation and fluttering at the precordium (*vide* "Neurosis," Mr. Ralehan); variation in the rate of the pulse without irregularity; and flushings. There is likewise usually anæmia in some degree, and, in young females, leucorrhœa and cervical venous hum.

2nd. Attenuation and weakness of the ventricles, as exhibited in middle age by those of a nervous and fretful temperament, and evinced by feeble cardiac action with or without irregularity, and sharp clear sounds.

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* *Ludwig's Arbeiten*, 1868. Abstract by Hermann in *Centralblatt*, No 31, 1868; and *Dublin Quarterly Journal of Medicine*, November, 1869.

† *Virchow's Arch*, xlvii, p. 223, also *Journal of Anat. and Physiology*, No. V, (November, 1869), p 168.

3rd. Weak and softened heart in process of tissue-degenera-
tion, with dilated and atheromatous arteries.

4th. Ventricular hypertrophy of the *simple* form.

The attempt to explain the phenomenon of reduplication of
the first sound, in any of these conditions, would involve specu-
lation in the region of the unknown. In the first three there is,
presumably, derangement either of synchroniety in the influx of
nerve-force, or of muscular irritability in the walls of the heart.
It is conceivable that, owing to one or both of these causes, the
contraction of the papillary muscles, and, therefore, the tension
of the tendinous chords and the valves, may be perceptibly later
in occurrence than that of the ventricular walls. Such derange-
ment would not, of necessity, involve retroversion of the valves
and reflux of blood, because, by the pressure of the mass of
blood, the valves are brought into mutual apposition at the first
moment of ventricular contraction, whereas, valve-tension and
sound are the joint product of parietal and papillary contrac-
tion. In the former of these periods the active pressure upon
the valves is comparatively insignificant, and the mere exten-
sion of the papillary muscles and tendinous chords suffices to
prevent their retroversion ; but, during the latter period, cor-
responding to the acme of ventricular systole, the active con-
traction of the papillary muscles is requisite to obviate that
result. Of its occurrence in simple hypertrophy, an explanation
will be given further on.

Reduplication of the first sound I therefore regard as equi-
valent to the resolution of it into its two constituent elements,
the ventricular impulse, and the click of valvular tension ;
accordingly, the former element of the double sound should be
of a dull and muffled character, and the latter sharp and clear.
Such is actually the case, according to my experience, without
an exception.

Suppression of the first sound may occur in either of the
following conditions, viz:

1. Adipose softening of the heart.

2. Typhous softening of the ventricles.

Loss of the first sound, in the former of these conditions, is the
result in greatest part of the enfeeblement of the ventricles by

structural deterioration, by which both the impulse of the ventricles and the tension of the valves are impaired; it is, however, in some degree likewise due to imperfect conduction of sound through the softened walls of the ventricles. It will be hereafter shown that suppression of impulse and of first sound, in association with a certain group of symptoms having reference chiefly to the encephalon and the pulse, warrants the diagnosis of fatty heart.

Louis has called attention to suppression of the first sound of the heart in enteric fever, as indicative of softening of the ventricles.[*] Dr. Stokes subsequently pointed out this sign as peculiar to typhus fever from the sixth to the fourteenth day; and in typhus, and when not previously exhibited, as pathognomonic of ventricular softening. Most frequently the left ventricle only will be found in this condition; occasionally both ventricles are engaged, but the left in a greater degree than the right; and still more rarely the entire organ is similarly affected. The condition adverted to is not the result of putrefactive change; it is strictly pathological, non-inflammatory, and specific in character. Although indicative of so grave a structural lesion of a vital organ, this sign is not usually associated with a fatal issue of the disease, and constitutes an indication for the free administration of wine.[†]

Association of phenomena with the systole of the ventricles. Concurrently with the commencement of ventricular systole, as already stated, the impulse and the first sound are delivered, which imply the impact of the apex against the chest wall, and the closure and tension of the auriculo-ventricular valves.

Preparatory to the impulse, as pointed out especially by Professor Halford,[‡] the base of the ventricles descends, whilst the apex ascends, but to a less extent, and the central portion expands or attains increased bulk. By the descent of the ventricles space is afforded for the diastole of the auricles, and a point of resistance is given to the aorta and pulmonary artery, which are about to be put upon the stretch by the vigorous

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* *Researches on Gastro-Enteritis*, Bowditch's translation, vol. i.

† *Dublin Journal of Medical Science*, and *Diseases of the Heart and Aorta*, p. 370.

‡ *The Action and Sounds of the Heart*, 1860.

injection of blood from the ventricles. The blood is driven with great force into the aorta and pulmonary artery, which are distended to their uttermost capacity, and the branches arising from the former of these vessels are filled consecutively in the order of their origin from the main trunk. Thus, the carotid and subclavian arteries are supplied and pulsate almost simultaneously with the impulse of the heart, but the radials perceptibly later than the impulse.

Dr. Burdon Sanderson says the radial pulse is one-sixth of a second later than that of the carotids, owing to the elasticity of the large arteries; where these are unyielding there is no postponement of the peripheral expansion. He says the heart exerts its greatest vigour at the commencement of ventricular systole, in forcing open the sigmoid valves. The rate of transmission of the percussion wave in the arteries he calculates at about 90 feet per second. The bursting open of the aortic valves causes a sensible vibration, which may be mistaken for ordinary arterial expansion (?), but it reaches the peripheral arteries in about 1-50th of a second.*

During the contraction of the left ventricle, according to Sibson, " the cavity changes its place, being more to the right, and with its axis, which was formerly in the direction of the auricle, now pointing to the aorta. The contraction proceeds in a twisting manner; the blood is, as it were, wrung out of the cavity, and with a current that takes naturally the twisted direction of the spring and arch of the aorta.†

" The cavity of the ventricle, which at the end of the diastole is somewhat egg-shaped, is totally changed in form at the end of the systole. It is then narrow and triangular, except towards the apex, where it is obliterated; and it presents a spiral twist, in a direction from the aortic orifice to the apex. This spiral course commences at the aortic valves, where it bears backwards and from right to left, is just appreciable in the body of the cavity, and becomes again marked towards the apex, where it bears forwards and from left to right."‡

* *Lectures delivered at University College*, Lecture vi. ; *Medical Times and Gazette*, April 29, 1871.
† *Prov. Med. Transact.*, 1844, p. 518.
‡ *Medical Anatomy*, last fasciculus, " Movements and Sounds of the Heart."

The blood thus escapes from the ventricle in a succession of spiral curves, by which, as in the rifle, precision of movement is insured, and the effects of friction are, to a certain extent, obviated.   The spiral fibres of the walls grasp the papillary muscles and fleshy columns "like the coils of a serpent," forcing them together and expelling the blood.   The mitral portion of the ventricle is entirely obliterated by the packing of the papillary muscles and fleshy columns, leaving for the passage of the blood only the small spiral channel already described, as leading in the direction of the aorta.

In relation to the closure of the mitral valve in systole, a space of much interest has been described by Sibson, under the name of "intervalvular space," into which "the aortic valves, during diastole, play directly;" and owing to the existence of which "the mitral valve is closed up to the end of systole, by the pressure of the blood on its anterior flap." "Its immediate walls are everywhere rigid and aponeurotic, and it cannot, therefore, be compressed by the contraction of the muscular walls during the systole."   This space is described as being bounded in front by the aortic valves, behind by the anterior segment of the mitral valve, to the right by the central fibro-cartilage or tendinous septum, and externally or to the left by the muscular wall of the ventricle, "which is here lined by a rigid aponeurosis."*

He says that, in contraction, the left ventricle forms a shoulder round the mitral valve, which, however, it never touches.   The direction and range of movement of the walls of the several chambers of the heart, and the relative direction and course of the several currents of entrance and exit, are admirably represented in the annexed engravings (Figs. XVII and XVIII), borrowed from Dr. Sibson.

Dr. Sibson refers to these engravings in the following terms: " In order that the successive movements of the heart might be observed with accuracy, a millimetre measure was stretched across the organ, as in Fig I [XVII], and its precise position in relation to the measure was noticed, first at the end of the diastole, and then of the systole." "In those figures [XVII and XVIII] the

* *Medical Anatomy.*

FIG. XVII.

FIG. XVIII.
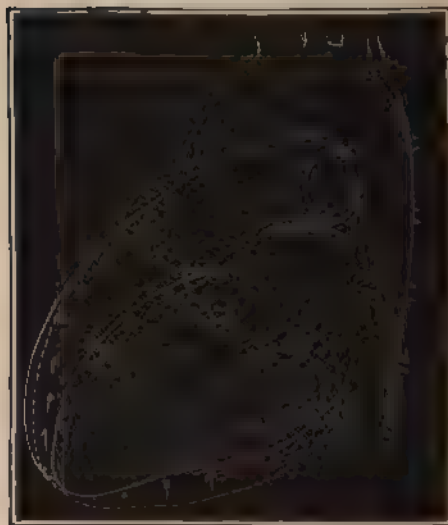
Fig. XIX.   Fig. XX.
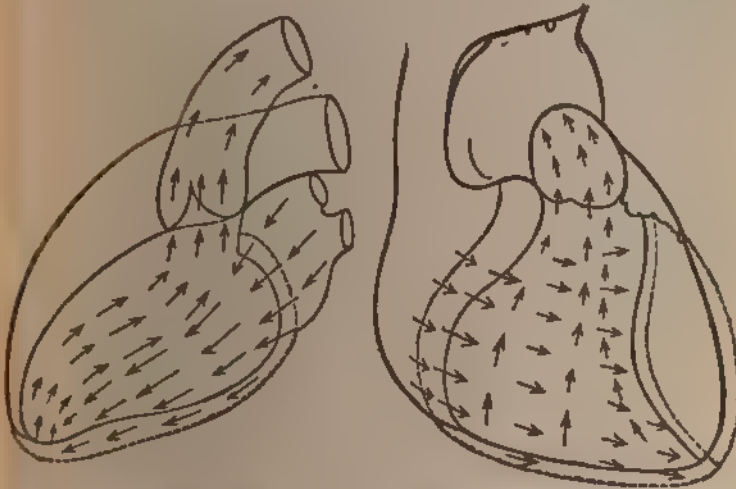
outlines of the cavities of the heart and the great vessels are in-
dicated, at the end of the systole of the heart by continuous lines;
at the end of the diastole by interrupted or dotted lines. The
dotted are less accurate than the interrupted lines. The systolic
movements are everywhere shown by arrows, the extent of the
movements being marked by the length of the arrows. It is
more difficult to watch and measure the movements of the heart
at the side than at the front. The side view, Fig. II [XVIII], is,
therefore, not so accurate as the front view, Fig. I [XVII]. I
believe, however, that both figures very fairly represent the
movements of the heart.

"In the left ventricle, the aperture of entrance through the
mitral orifice is contiguous to the aperture of exit at the aorta,
the two orifices being separated by a membranous septum. In
the right ventricle the aperture of entrance through the tricuspid
orifice is at a distance from the aperture of exit at the pulmo-
nary artery, the two orifices being separated by the muscular
channel of the *conus arteriosus*. In the left ventricle the current
of blood inwards, which descends during diastole behind the
anterior segment of the mitral valve, is parallel in direction to the

current of blood outwards, which ascends during the systole in front of that segment [Fig. XIX annexed]. In the right ventricle the current of blood inwards is at right angles to the current of blood outwards, since the blood enters the cavity from right to left, and leaves it from below upwards [Fig. XX annexed]. During the systole the stream of blood in the left ventricle takes a spiral direction towards the aortic orifice, in accordance with the spiral direction of the aorta itself [Fig. XIX] The stream of blood in the right ventricle, as it ascends, mounts over the bulging septum, being restrained by the concave free walls. This upward stream, which narrows as it proceeds, thus takes the curved direction upwards, backwards, and inwards, of the *conus arteriosus* and the pulmonary artery. In the left ventricle the anterior segment of the mitral valve, and the right and left papillary muscles, form a hollow channel for the stream of blood, which, as it ascends to the aorta, presses upon the under surface of the valve. In the right ventricle the stream of blood, as it ascends, sweeps onwards at right angles to the under surface of the tricuspid valve, and rushes between and across the papillary muscles, and through the tendinous cordage that connects those muscles to the valve."[*]

I am convinced that the relative position of the papillary muscles and fleshy columns on the inner surface of the ventricle is such that, at the acme of systole, the former pass mutually to opposite sides of the chamber, and so by crossing lines of tension stretch the valves to the uttermost, and thus oppose to the escaping blood at the acme of systole, the maximum of resistance.[†] Sibson says the tip of the anterior right papillary muscle contracts towards the septum, and those of the left approach each other, and both extremities are approximated to the " zone of rest, or stable equilibrium."

The short, or systolic pause, equal in duration to one-third of the systole, immediately succeeds the impulse and the first sound. During this brief period of silence the ventricles are still in contraction, and blood is in forced and rapid efflux through the aorta and pulmonary artery, the auriculo-ventri-

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[*] *Opus citat.*, p. 18.

[†] See a paper by the author in the *British Medical Journal*, December, 1867.

cular valves being closed, and the sigmoid valves raised. If the ventricles were not still in contraction, the second sound, being determined by arterial reaction, would immediately succeed the first. On the other hand, it is no less clear that if ventricular systole persist through the short pause, the first sound, if in any measure due to the mutual friction of the fibres of the ventricles in contraction, or to the attrition of the escaping blood against the walls of the ventricles and arteries, should be likewise, in some degree, continued through it. That no sound is audible, however, through one-third of the period of ventricular systole, is to my mind proof that neither of these alleged causes of the first sound contributes in any measure to its production.

The force actually exercised by the heart in contraction is a problem of great interest, but the solution of it is attended with much difficulty, partly from the abstruse nature of the inquiry itself, but in a great degree also from the number of minor problems involved in it.

Hales calculated from his experiments that the hydrostatical pressure within the hearts of oxen and horses amounts to nine feet perpendicular of liquid blood.

Dr. Haughton estimates that in the human heart at 9·923 feet, and calculates that each ounce of human heart-muscle is capable of lifting a weight of 20lbs. through the height of one foot every minute; and finally, that in health, a propelling force equivalent to this is actually in operation throughout our lives.*

The *second sound* terminates the short or systolic pause; it is sharp, clear, and abrupt; loudest at mid-sternum, and at the sternal end of the left second intercostal space. Around each of these centres it is diffused through a radius of about two inches in the adult male, but with greater distinctness in three lines of direction than in others, viz, downwards and to the left towards the apex; vertically downwards to the xiphoid cartilage; and upwards to the right second costo-sternal junction.

The second sound is much more extensively diffused in females and in children, because in childhood and in the female sex it is of a sharper and clearer quality, and the thoracic frame-

* *Lectures on The Principle of Least Action in Nature.* Lecture III. ; *British Medical Journal,* June 17, 1871.

work in these subjects is a better conductor of sound. I have repeatedly heard the second sound, and the first still more distinctly, under both clavicles, and extensively on either side of the back, in nervous females. In children this is the rule, and absence of the cardiac sounds at any point of the chest, the rare exception.

As to the *cause* of the second sound, there is now no second opinion amongst writers of authority. It is the product of the sudden closure and tension of the sigmoid valves in the aorta and pulmonary artery, under the elastic reaction of the walls of these vessels, consecutive to ventricular systole. The contractile force of the ventricles is in great part stored up or rendered latent in the walls of these vessels, to be restored as reacting force, by which the vessels resume their normal diameter at the end of ventricular systole, propelling the contained blood onwards, and so completing and sustaining the wave of pulsation; but likewise repelling it in the direction of the ventricles, and thus closing the sigmoid valves with an audible click, constituting the second sound. The two sounds of the heart may be therefore contrasted in regard to quality, duration, constitution, cause, and mode of origin as follows, viz. :—

		First sound.	*Second sound.*
Quality	.	Dull	Clear.
Duration		Prolonged.	Abrupt.
Constitution	..	Compound.	Simple.
Cause	.	Impulse, valve-tension (in part active).	Valve-tension (entirely passive).
Mode of Origin	.	Direct and immediate result of ventricular contraction.	Indirect and remote result of ventricular contraction.

Reduplication of the second sound is of much more frequent occurrence than reduplication of the first. It is most frequently met with in association with presystolic murmur, and may be therefore regarded as a *presumptive* sign of mitral obstruction.

Next in frequency of occurrence is the combination of double second sound with valvular inadequacy at the aortic orifice. In all such cases there is, of course, a murmur in association with one of the two elements of the double second sound, and this element is invariably the last. (Cases of Hutchinson, and Mrs.

Ewell.)*   The existence of a murmur of aortic reflux in union with the second element, renders it easy to identify this latter as aortic in origin.   In all such cases the left ventricle will be found dilated ; hence the evacuation of that chamber occupies a longer period, and the reaction of the aorta is postponed in an equal ratio, and rendered posterior in time to the corresponding phenomenon on the right side of the heart.   Conversely, when reduplicated second sound exists in connection with disease at either arterial orifice, it may be regarded as evidence of dilatation of the corresponding ventricle.   The cause of reduplication of the second sound is not, as has been alleged, derangement of synchronism in the closure of the segments of either sigmoid valve.   If such were the cause there should be a murmur of reflux always associated with the reduplicated sound, but this is notoriously not the case in the majority of examples of it.   Again, I have repeatedly made the following observation, which seems conclusive as against the alleged univalvular origin of double second sound   Whilst a double second sound was equally audible at the orifice of the aorta and that of the pulmonary artery, but most distinctly so at a point intermediate to and equidistant from both, a single second sound only existed at the distance of an inch or so external to either orifice in the horizontal line. Manifestly, if derangement of synchronism in the closure of the segments of either set of valves had been the cause of reduplication, the latter would have been audible to an equal distance from the orifice in the directions inwards and outwards, and it would have been most distinct at the orifice itself, and not midway between the two orifices.   The crucial test, however, is supplied where diastolic murmur exists at the aortic orifice ; in that case a second sound *and* a murmur are audible in the situation of both orifices, and at all points intermediate to them ; but over that of the aorta the murmur preponderates, and is posterior in time, whilst the normal sound is in excess over the valves of the pulmonary artery.

Where the second sound is reduplicated in connection with mitral stenosis, the element deranged is most likely that produced in the pulmonary artery, the entire pulmonary system,

* *Vide* " Aortic Regurgitation " and " Mitral Obstruction."

and the right chambers being engorged by obstruction at the mitral orifice. In the effort to overcome this obstruction, the systole of the right ventricle is protracted, and the reaction of the pulmonary artery proportionately postponed. The reaction of the aorta is, on the other hand, in mitral stenosis most probably anticipated, where the left ventricle is reduced in capacity, as always is the case where mitral or aortic reflux does not co-exist. Hence, it is likely that in simple mitral stenosis two causes of doubling of the second sound are in operation; namely, diminished capacity of the left, and dilatation of the right ventricle.

I would, therefore, contrast the two conditions under which re-duplication of the second sound may be witnessed, as follows:

In Aorta.	*In Pulmonary Artery.*
1. Second sound, accompanied or replaced by a murmur, and postponed.	1. Second sound, normal as to time and quality.
2. Second sound, normal as to quality, but premature in occurrence.	2. Second sound postponed, but normal as to quality.

If the preceding observations be well founded, and I believe they are, it follows:

1. That dilatation of *either* ventricle must be accompanied by doubling of the second sound.

2. That the side of the heart at which the *postponed* element of the double second sound takes origin, is that of the dilated ventricle.

3. That equal dilatation of *both* ventricles will be unattended with doubling of the second sound.

4. That the arrest of reduplication of the second sound may be due either to improvement in the condition of a ventricle previously dilated, or to dilatation of a ventricle previously normal in capacity.

5. That, generally, the causes of reduplication of the second sound are those which give rise to difference in the capacity of the two ventricles.

I cannot agree with Dr. Richardson in the opinion that reduplication of the sounds of the heart is rare.* I have met with and noted it in seventeen instances: viz., in ten there was

* *Clinical Essays,* 1862.

reduplication of the first sound ; in six, of the second sound ; and in one, of both sounds.* Nor can I admit his assumption, to the effect, that a third short sound, taking the place of the pause, is frequently mistaken for a reduplicated sound. A short sound of this character, corresponding to what I shall have to describe as an "aborted" sound, is characteristic of failing heart, and *always* irregular in its recurrence ; whereas reduplication is singularly regular. He has not met with an example of doubling of the first sound, of which the majority of my cases are examples; and has met with only three cases of doubling of the second sound. His explanation of the phenomenon is, that it arises from asynchronism in the closure of the semilunar valves, consequent, as he believes, upon pulmonary obstruction, prolongation of the first sound, and retardation of the second sound on the *right side.*

Giegel and Guttemann coincide in the opinion just stated as to the cause of reduplication of the second sound. They have met with it pretty frequently in mitral narrowing, with or without regurgitation. Most of the patients were thin and anæmic, and had been long the subjects of heart-disease. The double sound was audible over the tricuspid, aortic, and pulmonary orifices. These writers regard doubling of the second sound, when constant, as diagnostic of stenosis or inadequacy of the mitral orifice.† It can, however, equally exist in all cases of stasis or engorgement of the right side of the heart, or of dilatation of the pulmonary artery ; but in these latter cases the doubling would not be so constant as where valve disease exists.

It may be also observed in young tuberculous subjects; in those suffering from pulmonary emphysema ; in pleurisy ; and in fatty degeneration of the heart.

Guttemann, however, does not agree with Geigel in the opinion, that doubling of the second sound is a *constant* sign of mitral constriction ; and in his dissent I fully concur. He regards the connection as rare, and present only when the patient

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* In several instances, not included in this summary, I have observed doubling of the second sound in connection with the dilated right ventricle of pulmonary emphysema, with intercurrent or chronic bronchitis.

† *Archives Générales de Medicine* June, 1869.

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is in a state of complete repose. This, however, is by much too exclusive.

Suppression of the second sound, I have witnessed only in the stage of collapse of Asiatic cholera. I believe I was the first to call attention to this negative phenomenon in cholera, in the report of the cholera epidemic of 1866, as witnessed in the Mater Misericordiæ Hospital, and published conjointly by myself and Dr. Cruise. Dr. Henry Kennedy of Dublin would seem to have observed it also in the same epidemic, as he announced his notice of it, and quite irrespectively of mine, in the discussion which followed the reading of the several cholera reports, at the College of Physicians in June, 1867. In the collapse of cholera the circulation is arrested primarily in the branches of the pulmonary artery, and retrogressively in the trunk of that vessel. Hence the twofold effect of *de*pletion of the left ventricle and aorta, and *re*pletion of the pulmonary artery and right ventricle. In the extreme of this condition it is conceivable how, from the opposite causes above mentioned, failure of re-action in the aorta and pulmonary artery, and therefore of the second sound, might ensue. Irrespective of the value of the foregoing hypothesis, and whatever the true explanation may be, the fact is unquestionable, that in the last stage of cholera, and, as far as I am aware, in that condition exclusively, complete suppression of the second sound may be observed, as of almost constant occurrence.

The observations of Dr. Stokes on the modification of the impulse and the sounds of the heart in typhus fever, are of the utmost value and importance, and will find a suitable place here.

A jerking impulse, with clear sounds, in the advanced stage of typhus, should be regarded with much apprehension of a fatal issue.

Partial or total loss of impulse, in connection with suppression of the first sound on the left side exclusively, or on both sides of the heart, has been repeatedly observed from the eleventh to the fourteenth day of typhus, but it has been met with as early as the seventh day. In these cases one or both sides of the heart, according to the extent of suppression, has been found

soft, friable, and livid, with a viscid exudation between the fibres. In one remarkable example (Case 48) not only the impulse, but both sounds also, were lost on the twelfth day of fever ; the heart was found to be soft and flabby, spreading out upon the table by its own weight, and when held up by the great vessels, resting on the closed hand, it fell over the latter like an inverted cup. The order of modification of the sounds, he has observed to be as follows :

1. Diminution of first sound over left ventricle.
2. Diminution of first sound over right ventricle.
3. Cessation of first sound over left ventricle.
4. Cessation of first sound over right ventricle.
5. Return of first sound over right ventricle.
6. Return of first sound over left ventricle.

He has observed, likewise, a fœtal character of both sounds, which, nevertheless, were distinct, the pulse being over 130; and he has remarked that in some cases of typhus where the first sound was lost, the radial pulse continued, and even exhibited average strength and volume. The radial pulse has continued thirty-six to forty-eight hours after all trace of impulse and sound had disappeared. From the seventh to the fourteenth day of a case of typhus the first sound and impulse were nearly lost, whilst the second sound was very loud, and a second and strong impulse existed.[*]

The diastole of the ventricles commences with the second sound, and terminates only with the next succeeding impulse and first sound. The cause of the initiation of diastole has been by some writers considered to be the impetus of the blood propelled into the ventricle by the auricle during the contraction of the latter cavity. In refutation of this doctrine it is sufficient to observe, that active contraction of the auricles takes place only at the *end* of ventricular diastole, and immediately previous to ventricular systole. Longet maintains that the movement of ventricular diastole is due to the two-fold cause of the elasticity of the walls of the ventricle, by which, after contraction, they tend to separate, and thus attain a state of equilibrium, as does a caoutchouc bottle ; and the influence of a virtual void in the

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[*] *Diseases of the Heart and Aorta,* p. 328, et sequent.

chest, created by the retraction of the lungs in expiration.* I believe the ventricles, especially the left, possess normally an intrinsic power of expansion in virtue of their elasticity, but I also hold that this intrinsic power is materially supplemented by the extrinsic force of auricular reaction and contraction, operating through the medium of a large mass of blood, the mere weight of which contributes in some degree to the expansion of the ventricles. It should be remembered that ventricular diastole *cannot* in health coincide with expiration oftener than once in every four pulsations of the heart, and it *may* not even so often. Dr. Garrod maintains the existence of an active diastole of the ventricles, dependent upon the active turgescence of the walls of the heart, " consequent on the flow of blood into the coronary arteries immediately after the systole," " the auricles, from their thinness, not being similarly affected." He endeavoured to prove this by injecting water backwards into the aorta of a sheep's heart. He further concludes that, during ventricular diastole, there is in operation an absorptive force or attraction towards the ventricles.† Independently of the objection that, under the influence of the same agency, the auricles are supposed to contract and the ventricles to dilate, it must be remembered that in Brackyn's experiments the alternate action of the auricles and the ventricles was maintained without a coronary circulation of any kind.

The duration of diastole is equal to two-thirds of the entire cycle, and it includes three important events; namely, the second sound previously discussed; the long pause; and the acme or momentum of auricular systole. (See Fig. XVI.)

The long or diastolic pause is equal in length to the diastole, *minus* the second sound. It is a pause only in the sense that no movement of the heart, audible or palpable through the walls of the chest, takes place during its continuance; but in no other.

At the commencement of diastole, and coincidently with the second sound, the auriculo-ventricular valves are thrown open, partly in consequence of the removal of pressure from their

* *Traité de Physiologie*, 3ième edition, 1869, tome ii. p. 132.
† *Journal of Anatomy and Physiology*, No. iv., May, 1869.

ventricular aspect, which occurs at the cessation of ventricular
systole, but mainly owing to the pressure on their auricular sur-
face, of the mass of blood which had been accumulating in the
auricles during the systole of the ventricles, and which now,
under the reaction of the auricular walls, forces open the valves
and enters the ventricles at a gush.

An extreme degree of mitral narrowing, or other form of ag-
gravated obstruction at the mitral orifice, may give rise to a
quasi mitral diastolic murmur, but such murmur is really post-
diastolic in time.[*]

A true apex diastolic murmur may arise from aneurism of
either ventricle of the heart, *and from this only*. Both the cause
and the phenomenon are of extreme rarity. I cannot recall
more than one example of the latter. (Case 68, James Toole.)[†]
Dr. Sibson is of opinion that the auriculo-ventricular valves are
raised in ventricular diastole by the papillary muscles, and
" when diastole is complete the anterior flap (of the mitral valve)
is held tight between the two papillary muscles."[‡] Notwith-
standing the high authority of Dr. Sibson I doubt the correct-
ness of this view.   I incline to think that if such were the
agency by which the auriculo-ventricular valves were raised in
diastole, and if, at its acme, they were thus held open and fixed
by unyielding tendons and extended muscles, at the first mo-
ment of ventricular systole reflux into the auricle and systolic
murmur would inevitably occur. On the contrary, I believe that
the papillary muscles and attached tendinous chords, in a state
of complete relaxation, and not hardened by immersion in spirit,
are of sufficient length to admit of the valves closing over the
orifice, even in the state of complete diastole of the ventricle.
Blood entering the ventricle from the auricle first displaces the
valve, pressing its segments against the walls of the ventricle ;
in proportion as the ventricle becomes filled, blood accumulates
between its walls and the ventricular surface of the valves, and
the latter are by this pressure displaced, and *floated* towards the
auriculo-ventricular orifice.   During this entire period, commen-
cing with the second sound, and extending through the greater
portion of the long pause, the auricles are in a state of undu-

* *Vide* " Mitral Obstruction."   Cases of Anne Coates and Mary Brennan.
  † *Vide* " Aneurism of Heart."   ‡ *Opus citat.*, p. 17.

latory or vermicular contraction, setting in from the appendix
in the direction of the ventricle. By this movement of the auri-
cles, the blood which enters them from the great veins is passed
as quickly into the ventricles, but without the pressure of active
muscular contraction. At the close of diastole, however, and
therefore at the end of the long pause, the auricles having at-
tained, in the language of Cruveilhier, a *momentum*, or condition
of nerve-force most favourable to contraction, compress their con-
tents with vigour, and propel the blood into the ventricles. These
chambers, being thereby brought into a state of maximum disten-
sion, and receiving the stimulus to active contraction which that
state supplies, contract in their turn, and so complete the cycle
by initiating another act of ventricular systole. The long pause
therefore commences after the second sound, and terminates at
the first sound ; and the movement of active auricular contrac-
tion which I have designated the *momentum of auricular systole*,
concludes the long pause, and completes the diastole and disten-
sion of the ventricles. (See Fig. XVI.) It is readily conceivable
that obstruction to the entrance of blood from the auricle would,
at this period, when the other condition necessary for the pro-
duction of bruit is supplied, namely, maximum pressure from
behind, be most likely to give rise to murmur; and such is ac-
tually the case, as witnessed in the production of the pathogno-
monic sign of presystolic bruit.

Longet admits the two pauses, and describes the first "as a
slight interval between the contraction of the auricles and that
of the ventricles;" and in reference to the second he says, "after
contraction of the ventricles the entire heart is relaxed or in a
state of repose, during which it fills."

In regard to the relative effect on the two pauses, of variation
in the rate of pulsation, Dr. Burdon Sanderson remarks :* "The
closure of the aortic valve was followed by a diastolic wave of
great intensity, with reference to which it was interesting to
notice that in this, as in all other instances in which I have had
occasion to make the observation, the pyrexial acceleration of
the pulse was not attended with any change in the duration of

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* " A Lecture on the Characters of the Arterial Pulse, &c.," part ii. . *British
Medical Journal*, 20th July, 1867.

the systolic period, that is to say, the heart beat more frequently, not because its movements were more quickly performed, but it took a shorter time to repose between them."

In a paper read by me before the Medical Society of the College of Physicians of Ireland, in May, 1865, I stated as follows: " In regard to the effect of diminished rate of pulsation on the rhythm of the heart, it would appear that the diastolic pause is *relatively*, as well as absolutely prolonged thereby. I have had for some time past, and still have under observation in hospital, a case of permanently slow pulse; it is that of a man aged seventy-five, with œdema of the feet and other symptoms of weak heart. The pulse is perfectly regular, and in the recumbent posture only *thirty* in the minute. The heart's action is equally regular. The sounds are somewhat dull, but unattended with murmur. In this case, therefore, the heart pulsates once in every 2″, of which $1\frac{1}{2}$″ ($1\frac{1}{3}$″) less the duration of the second sound is occupied by the diastolic pause, and the remaining $\frac{1}{4}$″ ($\frac{2}{3}$″) by the first sound and systolic pause." "As to the connexion between the radial pulse and the movements of the heart, it is commonly supposed that the radial pulse succeeds the first sound, and comes between that and the second sound. This, however, is true only when the pulse is at or near its normal rate. When the rate is under 126 the stroke of the radial artery corresponds to the systolic pause; but if the pulse amounts to 126 in the minute, it will be found that the beat of the radial artery is shifted back in the order of phenomena, and is now contemporaneous with the second sound. If the heart's action be still further accelerated, it will be found that the radial pulse is postponed in the same proportion, and now falls within the period of the diastolic pause." *

I believe the opinion stated in the foregoing extract to be substantially correct; and with the qualifications as to time included within brackets, and without binding myself absolutely to the rate of pulsation given above, I still adhere to it in its entirety.

The pre-systolic contraction of the auricles is spoken of by Sibson as taking place "just before the ventricular systole,"†

* " On the Rhythm and Sounds of the Heart," *Dublin Quarterly Journal of Medicine*, (Vol. xl., No. lxxx, New Series.)

† *Opus citat.*, p. 4.

and Marey, in Fig. X. of his great work* represents the auricular systole by a rather abrupt ascent in the tracing, as preceding the systole of the ventricles by about one-tenth of a second. (See Fig. XXI.)

Accentuation of the second sound has been noticed in connection with three several conditions, viz. :

Disease at the mitral orifice,

Aneurism of the arch of the aorta,

Dilatation and atheroma of the arch of the aorta.

Skoda is of opinion that disease of the mitral valve, obstructive or regurgitative, is invariably accompanied by accentuation of the second sound in the pulmonary artery, and impairment or partial suppression of that sound in the aorta.† The explanation offered of this phenomenon is, that in such case the right ventricle, being of necessity hypertrophied, would, by its systole, dilate the pulmonary artery with increased force, and thereby induce a proportionate reaction in that vessel; whilst the volume of blood entering the aorta, being reduced, would dilate that vessel but imperfectly, and thereby induce feeble reaction and faint second sound in the aorta.

Manifestly the organic changes necessary for the production of these secondary phenomena are of late occurrence in mitral disease; nor is right ventricular hypertrophy always associated with it. In many cases the right ventricle is found to undergo simple dilation, without increased parietal development. These are cases in which tissue change of a degenerative character is in actual progress in the right ventricle, and the same cause which prevents hypertrophy, precludes likewise its consequence, accentuated second sound. Whatever the explanation may be, I am safe in asserting that not one-half the cases of indubitable disease of the mitral valve which have come under my notice, have presented the sign of accentuated second sound in the pulmonary artery. This sign is, however, manifested in very different numerical proportion in the two forms of mitral disease. It is much oftener associated with narrowing of the mitral orifice, than with inadequacy of its valves.

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* *Physiol. Médic. de la Circulation du Sang.*
† *On Auscultation*, by Markham, p 334.

FIG. XXI.

[From Marey, *Fig. 10.*]
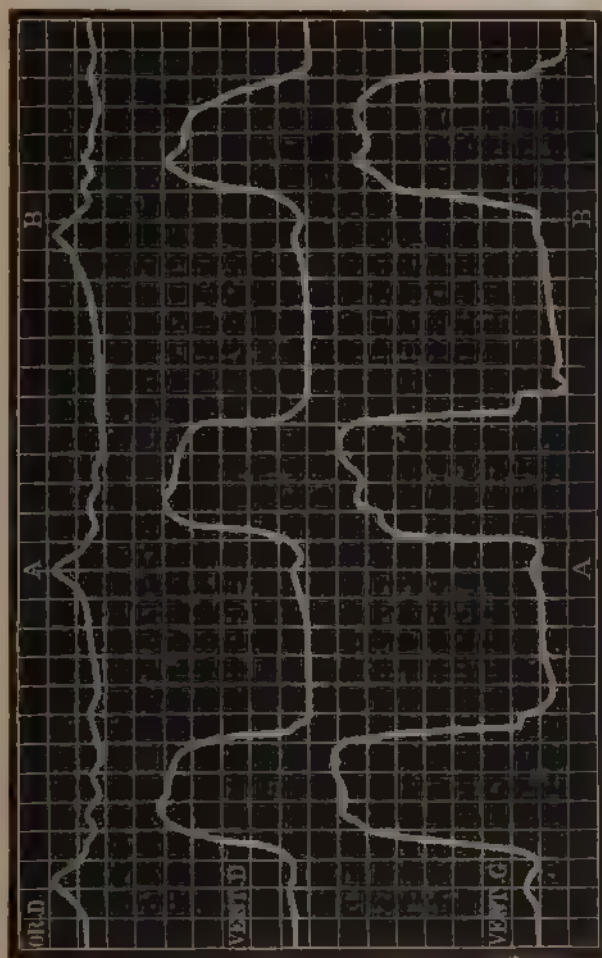
Dr. J. Warburton Begbie[*] calls attention to an " accentuated condition, or booming character" of the second sound, in aneurism, and in dilatation of the aorta ; he has found that, "excluding the accentuated pulmonary second sound, and the intensified aortic second sound in some cases of hypertrophy and dilatation of the left ventricle, the accentuated second sound in the aorta is an indication of aortic aneurism, or of dilatation of the aorta associated with atheromatous degeneration." "The aneurism probably does not arise within the pericardium, and probably does not affect the ascending portion of the arch, but has most likely its seat in the transverse portion."

He explains this by reference to "increased recoil of blood on the valves, owing to diminution or destruction of the support given to the circulation by the artery."

Morbid condition of the valves, thickening and patchy hardness of them, may heighten or intensify the sound ; and increased calibre of the vessel, may likewise, in Dr. Begbie's opinion, have the same effect.

Finally, he says that the sound is loudest over the aortic valves, both in aneurism and dilation of the aorta, but louder in the latter than in the former over the manubrium sterni.

Whilst fully admitting the deservedly high authority of Dr. Warburton Begbie on this and all other kindred subjects, I feel bound to declare that I cannot agree with him in the opinion, that accentuated aortic second sound is associated with aneurism of the arch, in sufficient numerical proportion to warrant its being regarded, even with the reservations mentioned, as a diagnostic sign of that disease. Its presence, in connection with thoracic aneurism, has certainly been, in my experience, the exception rather than the rule. The case is, however, different in regard to dilatation and atheroma of the aorta. In the absence of disorganization or inadequacy of the aortic valves, and associated with hypertrophy (with or without dilatation) of the left ventricle, a dilated and atheromatous condition of the ascending portion of the aortic arch is rarely unattended by accentuated second sound in the aorta. Nor is it difficult to explain the connection : the dilated and atheromatized vessel must con-

* *Edinburgh Medical Journal*, vol. viii., 1862-3

vey to the surface, more distinctly than in the healthy condition, the sound of valvular tension elicited by the reflected force of an hypertrophied ventricle. In cases of simple or dilated hypertrophy of the left ventricle, with inadequacy of the mitral valve, or with obstruction (with or without inadequacy) of the aortic valve, the inordinate contractile force of the left ventricle is neutralized by the valvular lesion; and similarly, the hypertrophy of chronic renal disease is most frequently rendered incompetent to elicit an exaggerated second sound by the consecutive advance of fatty degeneration of the ventricle. In the early stages, however, of the hypertrophy dependent upon Bright's disease, I have met with examples of intensified second sound; but I cannot recall a single instance in which atheromatous disease of the first portion of the aorta did not likewise exist.

The condition, therefore, in which this sign is most frequently met with, and of which it is in the great majority of cases diagnostic, is the two-fold condition of aortic atheroma and left ventricular hypertrophy; but, inasmuch as the former is usually the antecedent of the latter change, it may be regarded as, *par excellence*, the cause of, and the change indicated by, accentuated second sound in the aorta.

Transmission of the sounds and impulse of the heart is influenced by certain conditions of the intra-thoracic viscera which demand notice.

Laennec held that, " 1. Tuberculous excavation and pneumothorax transmit the sounds of the heart rather than its impulse, whilst hepatization and compression from effusion occasion results the reverse of these.[*] 2. The extent of pulsation (sounds) is in the direct ratio of the thinness and weakness of the heart, and consequently inversely as its thickness and strength. 3. The size of the organ is also favourable to the extent of pulsation, except where increase of size depends on thickness of walls exclusively. 4. The impulse is inversely as the extent of pulsation, and directly as the thickness of the walls of the ventricles. 5. Strong impulse is characteristic of hypertrophy. 6. The absence of impulse is characteristic of dilatation. 7. Hypertrophy with dilatation, the latter being in excess, communicates a

[*] *Forbes' Translation*, p. 540.

sensation like the blow of a mallet; it is not extensive, and does not elevate the wall of the chest, and the same result obtains, but in a less degree, in purely nervous palpitation. *
8. The softening of the substance of the heart renders the sounds much duller than natural." He adds, "In certain healthy subjects, in whom the walls of the heart are thinner than common, the contractions of the auricles are sometimes much louder than those of the ventricles below the clavicles, although the same disproportion is not observed in the cardiac region."

These propositions form a connected and well-ordered series, and I have arranged them in numerical succession for the purpose of more convenient reference.

No. 1 expresses an established and generally-admitted truth, respecting which it is unnecessary to use argument.

There is, however, one species of pleural effusion which possesses in a peculiar degree the property of transmitting the impulse of the heart, namely, empyema. Under certain circumstances, as yet unknown, purulent collections in the pleura exhibit in a high degree the property of vibration, in virtue of which they conduct and even intensify the pulsations of the heart. An example of this kind is given by Dr. Stokes; † the heart was displaced to the right; and the left side, which was the seat of empyema, pulsated throughout so as sensibly to shake the bed on which the patient lay. Yet the force of the heart's action seemed not greater than normal. Two cases of a similar kind, in the practice of Dr. Graves, are reported by Dr. Mac-Donnell. ‡

The extent to which the sounds of the heart are transmitted, irrespectively of the condition of adjacent organs or parts, is directly as the thinness of its walls, but *not* as the weakness of them. In an advanced state of fatty degeneration of its substance, the heart is often flabby, thin, and weak, yet the first sound is faint and ill-pronounced, and at a short distance from the superficial precordium, not at all audible; the second sound is likewise weak, and not extensively transmitted in the absence

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* *Loco citat.*, substance given, but not exact words.
† *Diseases of the Heart and Aorta*, p. 807.
‡ *Dublin Journal of Medical Science*, vol xxv., p. i.

of valvular lesion, unless the aorta be at the same time athero-matous, as usually is the case. With this exception the second proposition remains unchallenged.

Increase in the size of the heart, not due exclusively to thick-ening of its walls, implies absolute or relative attenuation. In the former case increased extent of pulsation will result, under the law expressed in the last proposition, reinforced in this case by the effect of increase in the volume of the heart.

In hypertrophy with dilatation, the condition in which the heart attains the greatest volume, the sounds are heard in front at a distance from the precordium, but they are of a masked or indistinct character; and posteriorly they are similarly diffused, owing to the extensive contact of the heart with the posterior thoracic wall.

The fourth and fifth propositions cannot be questioned, but although the absence of impulse (6th) is "characteristic" of dila-tation, it is by no means *pathognomonic* of that condition, as it is no less indicative of softening.

Proposition 7 must be admitted in its entirety, so likewise proposition 8; but the increased loudness of the second sound beneath the clavicles is due, I apprehend, not to thinning of the walls of the heart, but to increased density of those of the aorta, or to resistance in the pulmonary circulation. Again, the sub-jects of this remark may be simply nervous, and suffering from palpitation; the second sound in the aorta being sharp and ring-ing, as the result of an abrupt and forcible distention of the aorta, reflected upon the valves through the reaction of its walls.

The radial pulse, as the index of the state of the circulation, and remotely that of the nervous system, has been, from the ear-liest period of the history of medicine, assigned the foremost place in symptomatology. Recent investigations by means of the sphygmograph have invested this sign with importance, scarcely less than it possessed previously to the introduction of percussion and auscultation into practical medicine. By means of the radial pulse we may *approximately* determine the rapidity, force, and rate of contraction of the left ventricle, and by impli-cation, of the other chambers of the heart likewise. Certain forms of disease of the valves and of the substance of the heart,

and certain functional derangements of the nervous system, are also suggested by special characters of the radial pulse, as will be pointed out under the appropriate heads.

The state of tension or relaxation of the arteries, the volume of blood circulating within them, and the degree of resistance of the capillaries, are likewise indicated by the pulse at the wrist. Finally, the state and degree of nervous irritation; and impaired or suspended activity of the nerve centres, are indirectly represented in the radial pulse.

The rapid contraction of the left ventricle characteristic of the stage of reaction of all continued fevers, and of the hot stage of ague; of nervous excitement from depressing or exhilarating emotions; and from sharp but not exhausting pain; and of uræmic intoxication, is represented by an *abrupt pulsation of the radial artery*. Such is likewise the character of the pulse, but associated with weakness, and often with irregularity or intermittence, where the left ventricle is thin and dilated.

The beat of the radial artery is, on the contrary, *prolonged or drawn out* when the contraction of the left ventricle is sluggish or protracted. This happens especially in dilated hypertrophy of the left ventricle; it is also witnessed in apoplectic coma. In these conditions the pulse is likewise full, indicating a strong but not vigorous contraction of the ventricle; in the latter it is tense, but in the former yielding; a difference due to the vasomotor irritation of encephalic pressure. The strongest radial pulse is associated with left ventricular hypertrophy and dilatation, the hypertrophy being relatively much in excess; a condition frequently met with in the advanced stage of organic disease of the kidneys, especially of the cirrhotic form, and before tissue degeneration of the heart has supervened.

The *rate* at which the contraction of the left ventricle is repeated within a given time is ordinarily determined by registering the number of pulsations of the radial artery in the same period. There is, however, one important exception to this rule, namely, in the last stage of many diseases involving partial failure of the heart; notably in left ventricular dilatation without hypertrophy, in simple softening, and in fatty degeneration of the heart, the number of cardiac pulsations exceeds that of

the radial artery; the difference being due to the occurrence of certain *abortive* contractions of the ventricles, audible, and often even palpable at the precordium, but not represented at the wrist. The systole of the ventricle is blighted or inhibited in its progress, or its walls fail by simple debility to develop a force sufficient to propel the blood to the extremities. Such failure is usually irregular in its recurrence; but in at least one instance I have noticed it to be perfectly regular; an effective contraction of the ventricle, represented by a distinct pulsation at the wrist, being regularly succeeded by an abortive effort, cognizable only by auscultation of the precordium, and totally imperceptible in the radial artery. The patient in whom this remarkable phenomenon was presented was a young and delicate female, convalescent from enteric fever of a mild character, in whom the action of the heart was previously irregular and intermittent. There was no valvular disease, and the sounds were sharper than normal, indicating thinning of the ventricular wall. The alternation of effective and abortive pulsations of the heart continued for a period of twenty-four hours, the action then again became irregular and intermittent. After an unusually protracted convalescence the patient entirely recovered, the action of the heart becoming perfectly regular. This case furnished one of the most notable illustrations with which I have become acquainted, of the specific virtue of digitalis as a *regulator* of the heart's action. After one or two doses of the medicine, containing each five minims of the tincture of digitalis, the action of the heart became regular, and continued so as long as the digitalis was administered; but when it ran out, or was purposely suspended, the action became again irregular and intermittent, as was repeatedly witnessed by the class.

Infrequent pulsation of the radial artery, commonly but inappropriately designated " slow pulse," is especially characteristic of certain forms of fatty degeneration of the heart, as shown by Dr. Stokes,[*] who regards this phenomenon as indicative of "an equally weakened state of both ventricles."[†] Mr. Adams was the first, so far as I am aware, to notice the connection between

[*] *Dublin Journal of Medical Science, vol. xi., 1846.*
[†] *Diseases of the Heart and Aorta, p. 332.*

fatty heart and permanently-slow pulse.[*]   Dr. Henry Kennedy has likewise given special attention to this subject; and, in a memoir published in 1864,[†] expressed the opinion, that a pulse permanently under 40 is usually associated with the degenerative, as distinguished from the accumulative form of fatty heart.

I believe infrequent pulsation of the heart, in the circumstances mentioned, is directly due to impaired nutritive assimilation or malnutrition of the tissues, especially of the nerve-mass constituting the respiratory centre; a change probably due to arterial degeneration of the parts affected.   In such case inhibition of the heart, through the pneumogastric nerves, to the standard of impaired nutritive capacity of the tissues, would seem to follow, under the law of the organism by which functions, mutually dependent, exercise a reciprocally regulative influence.   Where vascular atheroma and softening of the respiratory centre had made progress, I should expect, in addition, slow and irregular breathing.[‡]

Excessive increase in the rate of arterial pulsation occurs most frequently in connection with organic disease of the heart; it is usually intermittent and paroxysmal, and associated with palpitation.   In one instance I have known the pulse to attain a rate of 210 in a minute.   The case was that of a young man (Case 78, Owen Gilson) suffering from mitral and aortic valve disease with considerable hypertrophy of the heart. Ordinarily, the pulse did not exceed 96; but during an accession of palpitation, to which he was subject without any assignable cause, the pulse usually went up to 198, and on one occasion, to 210 in the minute.   In accordance with the modern doctrine of pneumogastric inhibition of the heart, this phenomenon might be explained on the assumption that, owing to some unknown cause, the restraining influence of the pneumogastric upon the heart was suspended, and the organ consequently "ran riot."

The throbbing or collapsing pulse of permanent patency of the

* *Dublin Hospital Reports*, vol. iv., 1827.

† *Proceedings of Surgical Society*, March, 1864.

‡ I have not witnessed a pulse-rate under 28 in the minute.   Burns (*Observations on Diseases of the Heart*, 1809, p. 46) records a case of fatty and dilated heart, in which, at intervals, and accompaning paroxysms of dyspnœa, the pulse fell to *ten beats* in the minute.

aortic orifice exemplifies pulse of a special character, indicative of a particular form of valvular disease. It is, truly, "the pulse of an unfilled artery" striking the finger feebly and abruptly, and suddenly and completely subsiding. Pulse of this character is always *risible*; but so likewise is the pulse of tortuous and of rigid arteries, corresponding, respectively, to the early and the late stages of atheromatous change of the arteries in middle and advanced age. The devious bending of the artery, its slow distension and gradual subsidence, and the strength and firmness of the pulse in the former condition, serve to distinguish it from the collapsing pulse of aortic patency, which exhibits characters in all respects the reverse; and, in the latter, the rigid state of the vessel will alone suffice for the differential diagnosis.

Reduplication, or *dicrotism* of the pulse, is a phenomenon of much interest and diagnostic significance. It is met with under the triple combination of abrupt and feeble contraction of the left ventricle, imperfect distension of the aorta, and diminished capillary resistance. These conditions are supplied in a pre-eminent degree in typhoid or enteric fever, in which, accordingly, after the tenth day, I have rarely missed the phenomenon of dicrotic pulse. It is not, however, peculiar to, or pathognomonic of, typhoid fever. I have noticed it in convalescence from other wasting diseases, especially rheumatism unattended with cardiac complication, and in the anæmic condition associated with lead-poisoning;[*] but in an especial manner, and much greater degree, it is associated with enteric fever in the stages of eruption and defervescence.[†] The pulse is soft and compressible, and the first beat, which represents the normal pulse, and coincides with, or immediately succeeds the systole of the ventricles, is the stronger of the two. The second beat follows[‡] by continuous succession; that is, there

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[*] *Vide* " Pulmonary Artery," lesions of, case of Hegarty.

[†] Dr. Waters (*Diseases of the Chest*, second edition, 1873) declares that in pneumonia, he has found the pulse invariably dicrotic.

[‡] Czermak has shown, by means of his *photosphygmograph*, that the pulse in the dorsal artery of the foot is 0 018 sec. later than that in the radial artery, and that the latter is later by 0 159 sec., and the pulse in the carotids by 0 087 sec. than the shoc of the heart. H. E. Weber estimated the rapidity of the pulse-wave at about 28 5 feet per second; but this must be distinguished from the velocity of the blood itself,

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seems to be no distinct interval between the first pulsation
of the artery and the second, the stage of retrocession of the
former passing, by a gentle curve, into that of ascent of the
latter.   The subjoined figures (XXII, XXIII, XXIV, XXV,
XXVI, XXVII, XXVIII, XXIX, and XXX), well represent
the phenomenon of dicrotism under different conditions.   The
first was obtained from a patient under my own care; the second
and third are borrowed from Lorain.*

Fig. XXII.
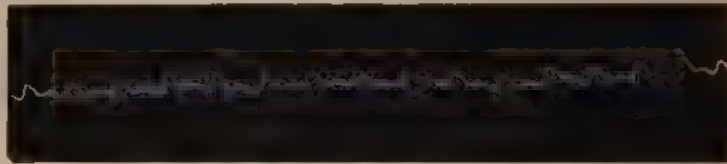
Typhoid (girl, Byrne 2th day), dicrotic pulse

Fig. XXIII.

Typhoid fever, hyperdicrotic pulse

Fig. XXIV
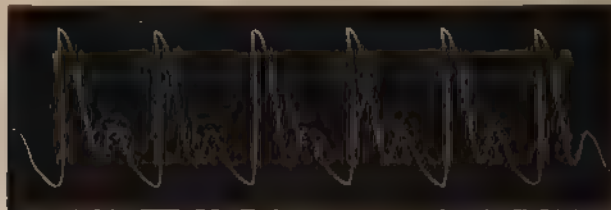
Acute rheumatism, dicrotic pulse.

Fig. XXX
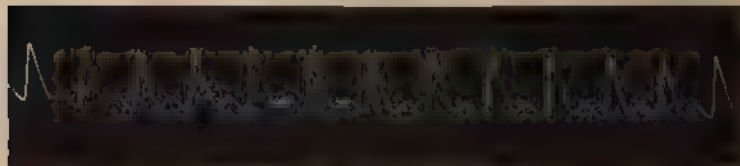
Dicrotism, not perceptible to touch, in a case of severe febricula

* *Etudes de Médecine Clinique*, 1870

Fig. XXVI.
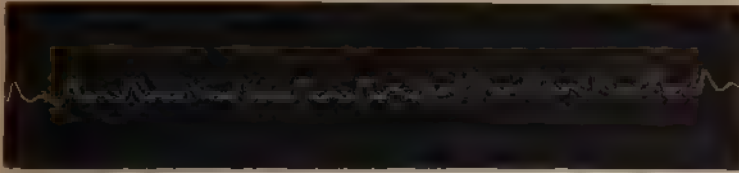
Dicrotism, perceptible to the finger, on the second day of slight febricula

Fig. XXVII.
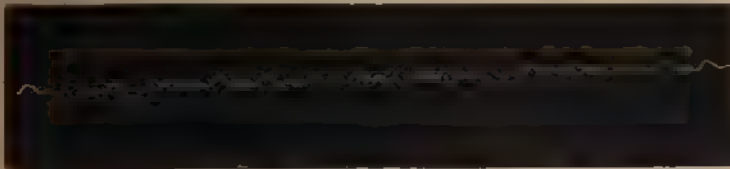
Dicrotism, not distinctly perceptible to touch.

Fig. XXVIII
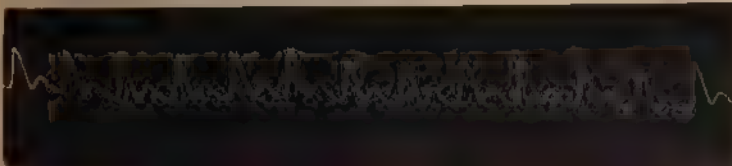
Distinct dicrotism, on the tenth day of typhoid fever complicated with pneumonia, and the day following profuse intestinal hæmorrhage. Several of the pulsations are hyperdicrotic.

Fig. XXIX.

Dicrotism, on the twelfth day of typhoid fever, quite perceptible to the finger

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Fig. XXX
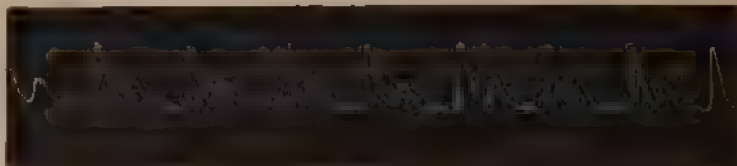
Dicrotism, perceptible to the touch, in a case of febrile excitement, accompanied
with diarrhœa, after a surfeit of unripe fruit.[*]

With few exceptions, authors are agreed in the opinion that
multiplication of the arterial pulse, proportionately to that of
the heart, is directly due to elasticity in the coats of the
arteries. Galen was the first to explain dicrotism by attribut-
ing it to vibration of the artery, produced by afflux of blood.
Vivenot (de Vienne) says, " The polycrotism is more pronounced
in proportion as the artery contains less blood relatively to its
calibre, and the more quickly it fills and empties itself." He
adds, that elasticity of the artery is likewise indispensable, as
exposure to compressed air arrests dicrotism.[†]

Ludwig regards the second pulse of dicrotism " as a conse-
quence of the elastic action of the first."[†]

Buisson and Naumann held that the first pulse was due to
systole of the left ventricle, and the second to recoil from the
sigmoid valves, of blood forced back towards the heart by the
systole of the aorta. Naumann satisfied himself, by experi-
ments with his artificial apparatus designed to represent the
heart, that dicrotism is incompatible with inadequacy of the
aortic valves ; for he found that, by partially or completely
destroying these, he could reduce or abolish dicrotism.[†] He
likewise found that dicrotism was more pronounced in propor-
tion to the proximity to the heart, of the artery examined, but
the undulations more numerous in proportion to its distance
from that organ.

In 1864 Landois also experimented with elastic tubes, and
confirmed the opinion of Naumann, with whom his results
placed him in strict accord.

* For the last six tracings in this list I am indebted to my friend, Dr. Grimshaw.
† Lorain, *opus citat.*, p. 34-47.

In regard to reduplication, this writer classified pulses under three heads; namely,

1. Those in which there is no reduplication, which he designates as "simple."
2. Those which are repeated in the line of ascent, "anacrotic."
3. Those which are repeated in the line of descent, "katacrotic."

Either of the two latter may be double or triple. Thus:

$$
\text{Anacrotic} \begin{cases} \text{Anadicrotic.} \\ \text{Anatricrotic.} \end{cases}
$$

$$
\text{Katacrotic} \begin{cases} \text{Katadicrotic.} \\ \text{Katatricrotic.} \end{cases}
$$

He held that, ordinarily, in the neck and adjacent regions the pulse was tricrotic.

Wolff found that a pulse incompletely dicrotic became completely so when the body temperature attained 31·8 to 32·4 Réaumur, and "superdicrotic" when a higher temperature was attained. In typhus he has found the pulse superdicrotic at all periods of the day.[*]

Marey regards dicrotism as a purely physical phenomenon, dependent upon two conditions; namely, velocity of current, and oscillation of the liquid column alternately from and towards the heart, the result of elasticity of the vessel.[†] He says "The oscillation which constitutes dicrotism occurs in the arteries of the periphery; this oscillation requires for its occurrence a rapid impulsion of the liquid, and a sufficiently large mass put in motion." He has succeeded in producing it in an elastic tube incompletely filled. The stronger the arterial tension, the greater is the resistance which the left ventricle encounters in propelling the blood into the aorta. If arterial tension falls below a certain degree, the left ventricle contracts with great rapidity, but without corresponding force, and the wave thereby caused travels with extreme velocity. But whilst defective arterial resistance, as shown by Marey, determines a premature arrival of the systolic wave in the peripheral arteries, it must,

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[*] Lorain (*loco citat.*), to whose exhaustive work I am indebted for the preceding references.

[†] *Physiologie Médicale, De la Circulation du Sang,* 1863, p. 201, *et suiv.*

by the imperfect tension of the aorta thence resulting, have the
opposite effect in regard to the wave of aortic reaction; namely,
that of postponing its arrival at the periphery.   Hence the
asynchronism of the ventricular and the aortic pulse-wave
which constitutes dicrotism.   Dicrotism is especially common
in the typhoid condition, and is of two kinds, which, however,
the finger cannot distinguish; viz., dicrotism of ascent, and di-
crotism of descent, of the pulsation-wave.   The latter, alone, is
true dicrotism.   He agrees with Bouillaud in holding that the
pulse is dicrotic in aortic valve inadequacy; but adds that it is
so in the line of ascent only.   In such case, dicrotism of ascent
is manifestly caused by obstruction at the orifice of the aorta,
and dependent upon the roughened, thickened, or tuberculated
state of the valves, which, in the majority of instances, is found
to accompany regurgitation by inadequacy of the valves.   By
way of explanation of the phenomena, he says, the jet of blood
from the left ventricle passes freely through the large arteries,
but is resisted in those of the periphery; from the latter there
is a reflux upon the aorta; but the aortic valves being closed,
another obstacle is presented to the reflux current at that
point, whence the blood "rebounds" in the direction forwards.
These oscillations continue till arrested by the succeeding con-
traction of the left ventricle.   He adds, "after the closure of
the sigmoid valves there occurs a new centrifugal current which
constitutes the secondary or dicrotic pulsation."[*]   This state-
ment was confirmed by the result of an experiment, consisting
in the application of the sphygmograph and Chauveau's regis-
tering hæmodromometer to the carotid artery of a horse pul-
sating dicrotously.   The statement and experiment just quoted
from Marey, show that he is substantially in agreement with
Naumann as to the cause of the second pulsation in dicro-
tism.   The rate of the circulation and the elasticity of the dis-
tal arteries exercise an important qualifying influence.   Thus,
if the blood arrive in the arteries slowly, there will be no
dicrotism; and with the same initial velocity, the peripheral
arteries being elastic there will be dicrotism, and being in-
elastic there will not be dicrotism.   Thus, in old subjects,

* *Opus citat*, p. 274.

owing to the loss of arterial elasticity, there is only imperfect dicrotism; in young persons, on the contrary, it is best pronounced; and in typhoid fever, in which it may be witnessed in the most typical form, the elasticity of the arteries is in excess, because their contractility is in defect. Arterial tension is associated with dicrotism; feeble tension favouring, and strong tension opposing it. Vertical posture is favourable to dicrotism in the arteries of the upper portion of the body, by lessening their tension. So likewise is intermittence of the heart, and for a similar reason, the beat immediately succeeding an intermission being more decidedly dicrotic. In the aorta and carotids of the horse there is always dicrotism, due to closure of the aortic valves; in the carotids of man there is likewise normally feeble dicrotism; but in the radials it is neutralized by the elasticity of the vessels.

The high authority of Marey upon this and all kindred subjects can alone justify the length of the preceding quotations from his work; and, whilst adopting his views in substance, I shall take the liberty of pointing out a few inconsistencies in them.

The alleged reflux of blood upon the aorta, from obstruction encountered in the peripheral arteries, should, of necessity, coincide with the initial period of ventricular diastole, and therefore with the systole of the aorta and the closure of the sigmoid valves; but the systole of the aorta determines a movement of the column of blood contained in it in two opposite directions; namely, towards the heart, by which the valves are shut down; and towards the periphery. The latter must, therefore, encounter and neutralize the alleged reflux from the peripheral arteries, and render it incapable of producing a secondary peripheral pulsation. The typical character of dicrotism in typhoid fever, and under similar conditions in the young subject as contrasted with the aged, I believe to be due, not wholly to a difference in the elastic quality of the arteries as such, but in a great measure to the different degree of resistance presented to the onward movement of the blood, to whatever cause due. I incline to the opinion of Wolff, already quoted, which implies that the dicrotism of typhoid is indirectly

due to the high temperature attained in that form of fever, by which peripheral resistance is lessened, rather than to that of Marey, who, adopting Weber's doctrine of the antagonism and reciprocally inverse manifestation of elasticity and contractility, attributes the dicrotism of typhoid to defect of contractility.

Dr. Burdon Sanderson's opinion on this subject may be collected from the following extract:

"This contraction (of left ventricle) produces expansion of the arteries, and acceleration of the progressive movement of their contents. These two associated and simultaneous effects are not only much more marked in the large arteries than in the small, but occur at an earlier moment, so that at the periphery the current attains its greatest acceleration *somewhat later* than near the heart.

"Hence, at the moment that the ventricle relaxes and the influx of blood through the aortic valve ceases, blood is still moving rapidly onward in the small arteries. As a necessary result, the arterial system becomes relaxed, and the progressive movement of the blood is retarded, collapse beginning where the stoppage occurred (viz., at the aortic valve,) and being propagated towards the periphery. Then, as the capillary arteries are relaxed, the capillary circulation is retarded, while the aorta becomes simultaneously distended in consequence of the increased resistance in front. This distension is, in its turn, propagated towards the periphery, and is succeeded, like the systolic distension, by collapse. If the conditions are favourable, the same series of movements may be several times repeated, the differences between the alternating conditions becoming less and less at each repetition. Thus, we have the following succession of phenomena:

"1. Contraction of the left ventricle.

"2. Distension of the aorta, and greatest acceleration of blood-stream in the great arteries.

"3. Greatest acceleration in the peripheral arteries, occurring simultaneously with cessation of progressive movement in the aorta.

"4. Diminished distension and diminished progressive movement in the aorta.

" 5. Propagation of these effects to the capillary circulation, and consequent increase of arterial resistance.

" 6. Propagation of the resulting aortic distension towards the periphery, producing peripheral acceleration ;"* and so on.

From the preceding extract, although the phraseology is not remarkably clear, it may be inferred that, in the opinion of the author, dicrotism is due to the vibration of elasticity, or alternate distension and reaction of the arteries. The word "collapse" is not strictly applicable to the aorta in the normal state of the circulation ; it applies only when the aortic valves are inadequate. In the preceding quotation it obviously means reaction ; but as reaction sets in progressively from the aorta towards the periphery, the blood must be propelled in advance of the reaction-wave, and hence there can be no obstruction to the aorta from retardation in the capillaries.

Reaction of the aorta immediately succeeds distension of that vessel, and is simultaneous with the commencement of ventricular diastole. As a consequence of reaction of the arch of the aorta, and proportionate diminution of its calibre, the blood-column which it contains is elongated both in the direction backwards towards the heart, and forwards towards the periphery ; the former of these movements is limited and suddenly arrested by the closure of the sigmoid-valves ; and, being still under the pressure of the reacting vessel, the retrograde current becomes progressive, in continuation of the primary reaction-wave.† With normal capillary resistance the action and the reaction-wave, or those of ventricular and aortic systole respectively, are in direct and unbroken continuity, and constitute, as

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* *Handbook of the Sphygmograph*, 1867, p. 23.

† I cannot admit the doctrine advocated by Longet (*Traité de Physiologie*, 5ième. edit., 1869; tome ii . p. 172), in the following words " Immediately after closure of the sigmoid valves, the blood which rests upon them is perfectly immovable till displaced by the next systole.' The aorta is elastic, quite to its root, and it is inconceivable that the portion of the vessel adjoining the valves, whilst possessing the same physical properties as the remainder of it, should be quiescent , all other portions being, at the same moment, in a state of energetic reaction. That the blood which rests on the aortic valves is capable of retrograde movement, is proved by the physical phenomena resulting from incompetency of the valves. Why, then, should it be incapable of movement forwards in the direction of the general current ?

judged by the tactile sense, but a single expansion of the artery.
When, however, the peripheral arteries and capillaries are
relaxed, or in a state of defective tonic resistance, the percus-
sion-wave flows off before aortic reaction can take effect; and
consequently before the flux of reaction, or tidal wave, setting
in from the aorta, can reach any of its branches. Hence double
pulsation or dicrotism.

If, in addition to premature escape of the flux of ventricular
systole in front, that of arterial systole be incomplete and tardy
owing to imperfect distension and feeble reaction of the aorta,
the interval between them will be proportionately longer, and
dicrotism more distinct in the arteries. Feeble contraction of
the left ventricle, by imperfectly distending the aorta, and
thereby inducing a weak and slowly-advancing tidal wave,
supplies this second condition. Hence it is that in typhoid
fever, a disease characterized by feeble circulation and nervous
exhaustion, dicrotism is most frequently and most typically pre-
sented. I can, to the full extent of my experience, confirm
Naumann's statement, that true dicrotism is never associated
with permanent patency of the aortic valves; and I therefore
incline to the opinion of Naumann and Marey, that a large
portion of the reaction-wave of normal circulation and of di-
crotism is the product of "recoil" or "rebound" from the closed
aortic valves.

Doctor Galabin maintains that the dicrotic wave is not solely
due to recoil from the aortic valves; for, as he alleges, "it
occurred when they were entirely absent." His experiments
were made upon elastic tubes attached to the heart of the sheep,
and to an artificial heart of india-rubber.* I am by no means
convinced by Dr. Galabin's experiments. It is notorious that
in the living body dicrotism of the pulse is *never* met with
where inadequacy of the aortic valves exists.

Dr. F. A. Mahomed adduces† the disappearance of the
dicrotic wave of the normal pulse-tracing, which occurs in
tracings obtained from the subjects of aneurism of the arch of
the aorta, as evidence of the proximal origin of dicrotism,

* *British Medical Journal*, September 6th, 1873.
† *Medical Times and Gazette*, August 9th, 1873

rather than of its distal origin as maintained by Dr. Burdon Sanderson.

Niemeyer regards dicrotism as a result of defective tonicity or sub-paralysis of the arterial walls.* But it should be remembered that, normally, the pulse is slightly dicrotic, although not perceptibly so to the finger.

Dr. Boileau agrees with Dr. Burdon Sanderson in attributing dicrotism to peripheral vascular resistance, although, with Niemeyer, he admits it may be in some degree due to defective arterial tonicity.†

* *Text Book of Practical Medicine*, 1869, vol. ii., p. 583.

† *Irish Hospital Gazette*, February 16th, 1874.

CHAPTER III.

PHYSICAL SIGNS OF DISEASE OF THE HEART.

HAVING now discussed the rhythm of the heart as a concrete phenomenon, and as such it certainly is the most striking manifestation of organic life, whether considered in regard to its regularity, its persistence, or its strict adaptation to the various and important purposes fulfilled by the circulation of the blood; I proceed to consider in this place, as furnishing the most appropriate connexion with which the subject may be introduced, the various derangements to which the rhythm of the heart is liable. These may be considered under five heads; viz., *intermission, irregularity, multiplication, suppression,* and *inequality* of the pulsations of the heart.

Intermissions of the heart have been classified by Laennec* as *true* and *false;* the former where the action of the heart is really interrupted; and the latter where it occasionally contracts so feebly that the wave does not reach the wrist; hence the radial pulse alone intermits in such cases. Bouillaud† maintains that these intermissions, whether true or false, are regular as regards their period of recurrence. I cannot agree with him in this opinion; on the contrary, intermissions, of whatever kind, are rarely rhythmical in recurrence. In one instance only have I met with rhythmical or periodically-recurring intermissions. A girl aged 22, of nervous and hysterical constitution, and the subject of obstinate abdominal pneumatosis, was received into hospital under my care in April, 1870. She was in apparently good health with the above exceptions; but the cardiac and radial pulse intermitted periodically, at intervals of a few seconds, the sounds of the heart being normal. Under treatment directed to the improvement of her general health, and

* *Traité de L'Auscultation Mediate,* tome ii., chap. viii.
† *Traité Clinique des Maladies du Cœur.*

especially of the nervous system, the pulse became quite regular, whilst the abdominal inflation continued unabated. I have not met with this symptom precursory to critical diarrhœa, as mentioned by Laennec.

True intermissions usually occur at short but unequal intervals of time; they are most frequently met with in old persons otherwise apparently healthy, and associated with a weak cardiac impulse, and sharp clear sounds; the arteries are likewise usually tortuous, and often atheromatous; in short, the symptoms and signs of dilated heart and atheromatous degeneration of the arterial system are usually associated with this form of intermission. It may likewise appear temporarily in convalescence from fever and other protracted and wasting diseases; and finally, in hypertrophy with softening of the heart. The length of the intermission may vary, as stated by Laennec, from one quarter to an entire pulsation of the heart; it usually succeeds the second sound. The existence of intermittence does not, of itself, warrant an unfavourable prognosis. I have known an old gentleman who, from early manhood, had been the subject of intermissions of this kind, and nevertheless, had gone through a laborious professional career, and lived to the age of eighty-one, dying ultimately of bronchitis and asthenia, but without exhibiting any of the ordinary consequences of disease of the heart. This gentleman's sister also died with similar symptoms at the age of seventy-six, and exhibited in her last illness intermitting pulse, with the signs of dilated heart and atheromatous degeneration of the vessels.

False intermissions may likewise occur in cases similar to the preceding, but are most frequently met with in connexion with the signs of simple dilatation of the heart, in persons, usually females, of middle age and weak constitution. Intermissions of this kind are occasionally witnessed in conjunction with irregularity, in simple functional derangement, the consequence of vicious habits.

I cannot subscribe the opinion expressed by Dr. Richardson in his otherwise admirable lecture* on intermittent pulse and

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* "Lectures on Experimental and Practical Medicine," *Medical Times and Gazette*, January 4th, 1868.

palpitation, that in the usual form of intermittency a second sound exists in the absence of a first; and that in the next pulsation succeeding an intermission the second sound is double; nor, assuming the existence of a double second sound as a usual concomitant of intermission, can I admit the explanation given of its occurrence, namely, suspended contraction of the left ventricle alone, whilst the right continues to act, and a consequent disproportion in the number, and asynchronism of the sounds produced by closure of the sigmoid valves on the two sides of the heart.

Separate contraction of one of the two ventricles, the other being contractile but quiescent, is scarcely conceivable, since their nerve-supply, and a considerable portion of their muscular walls, are common to both. No doubt, in the experiments of Schiff already mentioned (page 57), individual portions of the heart were paralysed by deligation of the branches of the coronary artery supplying them with arterial blood, whilst the remainder of the organ continued to act; but in this case contractility was annihilated in the part affected by the experiment, and the heart was thus reduced to the condition of a mutilated organ. In the hypothesis of Dr. Richardson, however, a general or constitutional cause, operating upon the entire nervous system, is supposed to induce *partial* paresis of the cardiac nerves, and paralysis of *one* of the four chambers of the heart. The explanation of reduplicated second sound offered by this author, would imply, moreover, that this phenomenon depends upon, or cannot be dissociated from, intermittence of the heart; but any such assumption is contradicted by the fact that reduplication of the second sound without intermission, is by no means uncommon, as the recorded examples of mitral constriction (see cases) will show. A faint first sound, followed by a proportionately indistinct second sound, as in the so-called false intermissions of Laennec, is of frequent occurrence, and quite intelligible; but a second sound, not preceded by ventricular systole, is to me incomprehensible. On the other hand, I have, though rarely, met with examples of intermission supervening immediately on the first, with loss of the second sound.

There may be *apparent* intermissions where two beats of the heart occur in rapid succession, followed by a prolonged pause, the prolongation, however, being in the direction *backwards;* that is to say, owing to the anticipation of the normal period of its occurrence by the preceding pulsation, the subsequent pause is proportionately prolonged. The apparent intermission is, therefore, no other than the normal long pause, with a prefix due to the premature occurrence of the preceding ventricular systole.

Irregularity is of much more frequent occurrence than intermission, though occasionally, and in certain cases, such as those of functional derangement, often associated with it.

Irregularity is, *par excellence,* the sign of cardiac neurosis ; and I quite agree with Dr. Stokes[*] in the opinion that, where associated with organic change, " it is more intimately connected with lesion of the muscles than of the valves of the heart." Hope[†] justly regards irregularity as of more serious import than intermission of the heart. This is, no doubt, due to the fact that it is more frequently associated with organic lesion ; and when it occurs in connection with valvular disease, there is invariably present likewise tissue change of the heart-substance, to which, rather than the valvular lesion, the irregularity owes its origin. In cases of acute disease of the valves, even though disorganization of their structure be already complete, irregularity is never witnessed unless when directly traceable to some other cause. Myocarditis, and pericarditis with serous effusion, usually give rise to irregular action of the heart. This symptom has been likewise observed by Graves as a precursor of pericarditis. It may occur in a transient form in connexion with gout and dyspepsia, and in an aggravated form, though amenable to sustaining and stimulant treatment, in what might be designated acute failure of nutrition in a weak and dilated heart. In all the foregoing examples of irregularity there is likewise inequality of the pulse ; that is to say, the beats are unequal not only in frequency, but likewise in force ;[‡] and there is no

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[*] *Diseases of the Heart and Aorta,* 1854, p. 175.
[†] *On the Diseases of the Heart,* 3rd edition, 1839, p. 377.
[‡] *" Faux-pas "* of Bouillaud.

standard proportion observed as between them in either of these respects. The irregularity is, therefore, *arythmical.*

There is, however, another form of irregularity in which the rate varies within a period of a few seconds; but the beats both of the heart and pulse are strictly proportionate to one another as to frequency and force, under each of these rates respectively. This form of irregularity, which may be distinguished from the preceding as *rythmical,* is strictly functional, and due to nervous derangement; it is of favourable augury, and according to my experience, exemplified only in cases of nervous debility consequent on masturbation, or the immoderate use of tobacco or of tea.

Dr. Richardson points out two forms in which irregularity of this kind, consisting in rapid changes in the rate of the pulse, may be exhibited. One he names "acute irregularity," in which the pulse beats at different rates in a series of five to ten or more pulsations; the members of the different series being strictly proportionate to one another in time, but differing in this respect from those of other series. This condition of pulse he has met with in cases of very feeble heart, in chronic anæmia, and after loss of blood or other serious depressing influence. The other form he names " prolonged irregularity," differing from the former only in this, that the series consist of a greater number of pulsations, and that it is best exemplified in the acute hydrocephalus of children, of which it is almost pathognomonic and of the worst augury.

I have never met with rhythmical irregularity of either kind under the circumstances mentioned by Dr. Richardson; indeed, in the conditions specified by him, I would rather expect to find examples of the first-mentioned, or arythmical form of derangement.

Inequality of the pulsations of the heart is always met with in association with false or apparent intermittence, of which, indeed, it constitutes an essential feature; and frequently likewise with arythmical irregularity, so often associated with the preceding derangements. Inequality in the action of the heart of necessity involves a similar anomaly of the pulse; and a case, by no means uncommon, in which all the foregoing derange-

ments are exemplified,* with the addition of a murmur, or perhaps more than one such, presents a jumble of sounds and movements of an apparently inextricable character, to be correctly interpreted only by those who, by patient study, have acquired a ready and precise knowledge of the normal rhythm of the heart.

Palpitation consists in rapid and tumultuous action of the heart, with or without derangement of rhythm, and painfully sensible to the patient. It comes on in paroxysms, during which the large arteries throb, and the nervous system is in a state of great excitement and tension. The attack is usually brought on by mental worry, bodily fatigue, dyspepsia associated with flatulence, or by late and heavy suppers ; but where organic disease of the heart exists it may occur irrespectively of any of these causes. Palpitation may be the disease ; that is to say, no organic lesion, so far as can be made out, co-existing with it. In such cases, which constitute a large majority of those in which it is met with, palpitation may be regarded as a veritable neurosis ; and the diagnosis rests mainly upon the negative evidence afforded by a physical examination of the heart, but in some measure likewise upon observation of the case, and the result of treatment. Thus, for example, it will be noticed that exercise in the open air and cheerful society produce a markedly beneficial influence upon the patient, warding off the attack which generally occurs at night, and insuring sound sleep and improved digestion. In many cases it is due to latent gout, and is readily cured by colchicum. The case of Mr. R.,† is a good example of palpitation due to this cause.

Palpitation may complicate organic disease of the heart, although it is present only in a small percentage of such cases. Dilatation of the left ventricle, with or without hypertrophy, and irrespectively of valvular lesion, constitutes the condition in which, *par excellence*, it is an associated symptom of organic disease. Attacks of this kind are much more protracted and obstinate than the preceding, the most efficacious treatment in the attack being antispasmodics, especially Hoffman's anodyne with

* *Folie des Battements du Cœur* (Bouillaud).
† *Vide* " Neuroses."

opium; and, in the intervals between the attacks, quinine, strychnine, and iron.   Case 68 appropriately illustrates these remarks.

One or other of the two sounds of the heart is not unfrequently found to be *reduplicated.* This occurs much more frequently in regard to the second than the first sound; although Hope, whilst admitting reduplication of the latter,* which he attributes to impulse of the apex out of time with the first sound, in which, according to him, it normally has no share, makes no allusion whatever to doubling of the second sound. Doctor Stokes† gives several examples of the latter, which he justly regards as the more frequent, and whilst confessing his inability to explain the phenomenon of reduplication, expresses the opinion that it is due rather to valvular than to muscular action, and is probably the result of a want of synchronism in the action of the two sides of the heart.

In regard to relative frequency of occurrence, reduplication of the second sound, according to my experience, greatly preponderates; of this several examples are given in the sequel, the great majority of which have occurred in connexion with obstruction at the mitral orifice.

Geigel and Guttemann state‡ that reduplication of the second sound has been pretty generally met with in connexion with mitral narrowing or inadequacy. Most of the patients were thin and anæmic, and chronic sufferers from heart-disease. The reduplication may be heard over the tricuspid, aortic, or pulmonary orifice, but is most distinct in the two latter situations; and at the site of the pulmonary opening the second of the two elements is louder than it is over the aorta, or is accentuated, in mitral valve-disease. They account for the phenomena by assuming derangement of synchronism in the closure of the two sets of sigmoid valves; the right ventricle being engorged in mitral disease, empties itself more slowly than the left; hence, say they, closure of the pulmonary sigmoid valve is posterior in time to closure of that of the aorta.

They regard doubling of the second sound, when *constant,* as diagnostic of stenosis or inadequacy at the mitral orifice.   It may

* *A Treatise on the Diseases of the Heart,* 3rd edition, p. 42.
† *Diseases of the Heart and Aorta,* p. 118.
‡ *Arch. Général de Médecine,* June, 1869.

exist also in stasis or engorgement of the right side of the heart, to whatever cause due; in cases of dilated pulmonary artery; in young tuberculous subjects; in the subjects of pulmonary emphysema, pleurisy, and fatty disease of the heart; but in these several cases they assert that it would not be constant as in mitral valve disease.

Guttemann does not agree with Geigel in the opinion that doubling of the second sound is an absolutely constant sign of mitral constriction, and present only when the patient is in a state of complete repose; he admits that when the heart's action is quickened the reduplication ceases. I agree with M Guttemann in the opinion that reduplication of the second sound is by no means constant in mitral constriction. It has occurred in only twenty-six out of sixty-three cases in my tables.

Mr. Arch. Bleloch[*] suggests that reduplication of the sounds of the heart may be due to interruption, in the cardiac plexus, of the wonted communication between the nerve-currents destined for the supply of the two sides of the heart. The simultaneousness of influx of the *vis nervosa* into the two sides of the heart would be thus prevented, and synchronous contraction consequently precluded.

He also offers, as another explanation, that whereas in man the *vis nervosa* travels at the rate of two hundred feet in a second, and in the frog only at the rate of ninety feet, neurosis of the heart may consist in "retrogression for the time being to a lower type of nerve-tissue;" *e.g.*, that of the frog. If, in consequence of unilateral neurosis, one side of the heart should undergo thus a slowing of its rate of action, "both the rate of transmission and the quantity of nerve-force transmitted may be less," and so derangement of synchronism of action take place. The first hypothesis is entirely gratuitous, being unsupported by evidence of any kind; and besides, it would not apply to examples of *temporary* reduplication. The second hypothesis, though exceedingly ingenious, is not only unproven, but in the present state of knowledge, not susceptible of proof or refutation.

Of reduplication of the first sound I have noted in all twelve cases; viz., eight females and four males. Of these six were

[*] *Medical Press and Circular,* June 22nd, 1870

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set down as "nervous," three being simply so with menstrual derangement; and of the three remaining, one (a male) was gouty, one (a female) was the subject of Graves' disease (exophthalmic goitre), and the third was an example of mitral constriction, both sounds being double. Hypertrophy of the left ventricle existed in three cases ; viz., in two with mitral reflux, and therefore with some degree of dilatation also ; the remaining example being one of simple hypertrophy consecutive to cirrhosis of the kidneys. Dilatation of the heart in connexion with cirrhosis of the liver existed in one case, and in two there was fatty degeneration of the heart.

I have met with reduplication of *both* sounds in two patients ; one, above referred to, was a female aged thirty years, and the subject of contracted mitral orifice ; and the other, a female aged seventy-five, suffering from bronchitis. In this latter case three sounds were audible at the base and at the apex respectively ; but in these two situations the doubled sound was not the same.

The causation of this phenomenon must be considered separately in regard to each of the sounds.

When associated with the first sound, I am satisfied it is due to resolution of that sound into its two normal elements, namely, the cardiac impulse, and the sudden tension of the auriculo-ventricular valves. In most of the cases it was possible to identify with great facility these two elements, the dull thud of the impulse having been, in every instance, antecedent to the sharp click of valvular tension. Cardiac impulse coincides with the initial portion of ventricular systole, whilst tension of the auriculo-ventricular valves and attached chordæ tendineæ occurs at the conclusion or acme of systole. Under ordinary circumstances the act is so rapid in accomplishment, that the two elements which enter into the first sound by which it is announced, are virtually simultaneous in occurrence. Not so, however, when any cause intervenes to protract the systole. In that case the initial portion of the first sound is dull, because due to the stroke of the apex only ; whilst the concluding portion, if the valves be in a condition to yield, the sound of tension on being suddenly put upon the stretch, is sharp and clear, because no longer masked by the dull element of impulse. In the event, however,

of the existence of valvular lesion competent to neutralize the sound of valve-tension, or to mask it by a coincident murmur, the impulse-element alone will represent the first sound, which, in such case, is necessarily of a dull or masked character, whilst a murmur, *post systolic* in time, represents the second element.

The organic lesions with which reduplicated first sound is associated are, in point of fact, of the above-mentioned character. Thus, of twelve cases in which this anomaly has been exhibited in my experience, four were examples of hypertrophy with dilatation of the left ventricle, and two of fatty heart. The condition of heart, however, with which, *par excellence*, reduplicated first sound is associated, is that of nervous instability, as exhibited in proneness to palpitation. Of this, no less than six of my cases are examples; but of these, one was also gouty, and one exhibited contraction of the mitral orifice and reduplication of both sounds.*

Doubling of the second sound is a phenomenon of a more distinctive character, and more frequent occurrence than the preceding. It is more easily appreciated because of the clear and decisive character of both its elements. It is due to derangement of synchronism in the closure of the two sets of semilunar valves, as the following observation, which I have repeatedly made, seems to prove. In every example of this anomaly which has come under my notice, the double character of the sound was exhibited only in the area over which the sounds of the aorta and the pulmonary artery were *both* audible; whereas, when the stethoscope was shifted a short distance to the right or left of this region, a single second sound only was heard.

Of twenty-seven examples which I have noted of reduplicated second sound, no less than twenty-six were cases of constricted mitral orifice, the remaining one being an example of aortic obstruction and reflux †

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* Reduplication of the first sound is of very frequent occurrence in simple hypertrophy of the left ventricle. In the cases above mentioned it would seem to be due to *post*ponement of tension click, and in simple hypertrophy to *præ*ponement of impulse.

† Between the dates at which the paragraph on this subject at p. 129 and the above were written, I had met with the additional examples of this phenomenon included in the number now given. Since the latter date I have noted several others, some of which will be found amongst the reported cases *passim*.

In the former cases it is not difficult to understand how, owing to imperfect distension of the left ventricle in diastole, and consequently of the aorta during ventricular systole, the reaction of this vessel, and the closure of its valves, shall occur prematurely, and be anterior in time to those of the pulmonary artery, and a double second sound be accordingly developed. The engorged state of the pulmonary artery consequent upon the obstruction in front, will contribute to the same result, by postponing the reaction of that vessel and the closure of its valves.

Where reduplication of the second sound is associated with valvular inadequacy at the orifice of the aorta, of even a few month's duration, the left ventricle may be assumed to be dilated. In such case the evacuation of that chamber is of necessity protracted, and in a still greater degree where obstruction at the mouth of the aorta coincides with permanent patency of the valves. Hence, the reaction of the aorta is delayed, and the aortic element of the second sound is dissociated from that of the pulmonary artery by postponement. Ultimately, however, owing to tissue-degeneration of its walls, the dilated left ventricle will fail to evacuate itself, whilst the right ventricle and the pulmonary system have become engorged. From this will follow the twofold result of an earlier reaction of the aorta, and a later reaction of the pulmonary artery. Unification of the second sound is the consequence of this pathological adjustment, and a double sound no longer exists. Such a result is obviously not to be expected, and in fact is never witnessed, in connexion with mitral stenosis.

Where mitral obstruction and permanent patency of the aortic valves co-exist, reduplication of the second sound will not occur, at least in the advanced stage of the twofold disease, when dilatation of both ventricles has ensued.

In Dr. Fuller's excellent work* there are some passages on this subject which I cannot subscribe. He says: " In all cases its [reduplication of sounds] essential cause is absence of synchronous action in the valves of either, or of both sides of the heart. Thus, if one ventricle contracts before the other, the auriculo-

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* *Diseases of the Chest.* p. 188.

ventricular valves on the two sides of the heart will not close simultaneously, and the first sound will be reduplicated; if the elasticity and irritability of the pulmonary artery and the aorta be unequal, the one vessel will probably contract before the other, and thus cause a want of synchronism in the closure of the semilunar valves on the two sides of the heart.

" But in some instances the reduplication appears to be confined to one side of the heart.

" In these cases there is *a want of synchronous action between the different segments of each set of valves;*[*] of the mitral or of the tricuspid, as the case may be, in the first instance; and of the aortic or of the pulmonary semilunar valves in the second instance."

In the preceding passages the valve-theory of the production of both sounds is exclusively adopted, and all other causes as contributory are excluded. This is not only not in accordance with, but directly contrary to, the bulk of evidence, experimental and clinical, on the point in question. As to the isolated contraction of either ventricle, upon which Dr. Fuller's theory rests, I have already given reason for doubting the possibility of its occurrence.

The aorta and the pulmonary artery are simply passive in regard to the circulation of the blood, and the time and force of their reaction depend not absolutely upon their elasticity, but upon the length and energy of the preceding contraction of the respective ventricles. The contractility of these vessels is, therefore, a borrowed property, and represents that of the corresponding ventricles.

As to the alleged want of synchronism in the closure of the different segments of each set of valves, adduced to account for the phenomenon of reduplication on *one* side of the heart, it is simply a speculation without basis or warrant of any kind, and opposed to everything that is known as to the function of the valves. Manifestly, the individual segments of any of the valves of the heart cannot be closed out of time with the others, and with a force sufficient to elicit from them a sound of tension, without being accompanied by a murmur of reflux; but it is

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* The italics are not in the original.

notorious that a murmur of any kind is a very rare accompaniment of a reduplicated sound at the same orifice.

Dr. Milner Fothergill regards the reduplication of the second sound as *prima facie* evidence of pulmonary engorgement, whether cardiac or not.[*]

But reduplication of the second sound is not even an exceptional symptom of simple pulmonary engorgement.

There must be, therefore, some other cause in operation where reduplication exists; and the association, which is undoubtedly frequent, of pulmonary congestion and double second sound, is the compound result of that common cause. (See p. 127.)

The phenomenon of *suppression* of either sound of the heart is strictly and exclusively morbid. *The first sound* is masked in hypertrophy, and all but extinguished in advanced fatty degeneration of the heart; and in typhus-softening, as pointed out by Dr. Stokes,[†] it is absolutely and entirely suppressed. The phenomenon is easily accounted for by reference to the morbid alteration in the muscular structure of the heart, in consequence of which, although sufficient to maintain a circulation adequate to the wants of the lowest degree of vitality, and barely to avert death, it is incapable of developing the energy required to produce impulse or systolic sound. In syncope, and as a precursor of death, failure of the first sound is a well-known symptom; and this sound may be masked or entirely superseded by a murmur.

Suppression of the second sound is of much less frequent occurrence than that of the first. As far as I know it is met with only in the collapse stage of cholera.[‡] It is probably due to extreme cardiac debility; but why, with an audible first sound, it should cease to exist in this disease, I cannot satisfactorily explain. Nor am I satisfied with the explanation of Dr. George Johnson,[§] who accounts for it on his favourite hypothesis of arrested pulmonary circulation; for although in such case the left ventricle, being deprived of blood, would be incapacitated for the production of a second sound, the right ventricle, for the opposite

[*] *Lancet,* November 6th, 1869.
[†] *Dublin Medical Journal,* vol. xv., 1839, p. 1.
[‡] See *Report of Cholera Epidemic* of 1866, by Drs. Hayden and Cruise.
[§] *British Medical Journal,* 1868.

reason, should be capable of producing one.   Yet nowhere in such cases is a second sound to be heard.   Both sounds are masked for obvious reasons in hydropericardium, and in pulmonary emphysema as usually met with, namely, that engaging the anterior edges of the lungs.   A condition the opposite of that just discussed, namely, that of exaggeration of the sounds of the heart, is of rather more frequent occurrence

Exaggerated first sound is met with most frequently in connexion with dilated left ventricle ; it is sharp, clear, and of brief duration, giving the impression of having originated in close proximity with the ear, and of having been transmitted through a thin and vibratile medium.   In extreme ventricular dilatation, when the heart has been excited to vigorous action by bodily exercise or mental disturbance, the quality of the two sounds is so nearly similar that it is exceedingly difficult to identify them individually.   But in dilatation and thinning of the left ventricle associated with tissue-softening, the first sound is abrupt, feeble, and usually ill-pronounced, and the action of the heart and pulse rapid and irregular, as pointed out by Dr. Stokes ;* and finally, in typhus fever similar phenomena are exhibited, as mentioned by the same authority,+ and both sounds not unfrequently assume a fœtal character, becoming weak and almost identical, whilst the action of the heart is exceedingly rapid. In the rare form of disease of the heart described by Dr. Ormerod‡ under the name of " universal fibrous transformation," the sounds of the heart are likewise of the fœtal character, and individually difficult of identification.

Exaggeration of the second sound is of more frequent occurrence in the pulmonary artery than in the aorta.   In the former of these vessels its occurrence is, according to Skoda,§ pathognomonic of mitral obstruction.|

My experience does not tend to confirm this statement.   I

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* *Dublin Medical Journal*, vol. xxi., 1842 ; *Pathological Society's Report*, p. 133.
+ *Ib.*, vol xv.
‡ *British Medical Journal*, August, 6th 1864.
§ *On Auscultation*, Markham's translation.
| Dr. W Begbie (*opus citat*) regards accentuated, or " booming " second sound in the aorta as suggestive of atheroma of the arch of the aorta, or of aneurism of its transverse portion.

have met with accentuated or intensified second sound in the pulmonary artery in only fifteen out of sixty-three cases, or less than one-fourth of the whole number of examples of narrowing or obstruction of the mitral opening; but in a large proportion of such cases, viz., twenty-six in sixty-three, as already stated, I have observed it reduplicated.

The extent to which the sounds of the heart, being normal, may be transmitted through the chest, depends upon many conditions having reference chiefly to the age of the subject, the configuration and conducting properties of the thoracic parietes, and the state of contiguous organs.

In infants and children, owing to the vibratile structure of the chest at this age, the sounds are transmitted to a greater distance, and with much greater clearness than in the subsequent periods of life. In those with narrow chests, especially of the pigeon-conformation, the sounds are likewise better transmitted, because of the absolute and persistent contact of the heart with the anterior thoracic wall.

Compressed, hepatized, or otherwise solidified lung, serves as a better conducting medium for the sounds of the heart than healthy lung substance.

Laennec asserts* that the sounds of the heart are better conducted than its impulse through tubercular vomicæ, and through the air of pneumothorax, whilst the reverse is the case in regard to compressed or hepatized lung.

Bouillaud holds that sound is transmitted through the latter no less distinctly than impulse. In this, I think, he is wrong. I apprehend that in all these cases alike the accompanying solidification is the cause of the exaggerated transmission, not only of the impulse, but of the sounds also. The walls of a tubercular cavity, being solid, and in immediate contact with air more or less stationary, are in the most favourable condition for transmitting sonorous and motor vibrations; and that motion should be better conducted than sound through a solid body, including in its interstices much liquid, such as a hepatized or compressed lung, will be readily understood. The pulsatile pneumonia of Dr. Graves affords a good illustration of this

* *The Diseases of the Chest*, Forbes' translation, 1827, p. 540, foot note.

remark.[*]   In this singular affection the throbbing of the chest must be regarded as an epi-phenomenon, dependent, according to Graves, upon the engorged state of the pulmonary vessels, which are thereby constituted good conductors of the cardiac pulsation.

Morbid alteration in the quality of the sounds of the heart, constituting *murmur*, has been, since the introduction of auscultation by Laennec, the subject of ardent and no less profitable study.   It is essentially a morbid phenomenon, and therefore appertains exclusively to the domain of pathology, but is not remote from the boundary line between health and disease.

A murmur may be *defined* as an abnormal sound developed within the heart, the blood-vessels, or an aneurism ; in the heart associated with, or superseding the normal sounds; in the arteries, in aneurisms, and in certain rare cases in the veins, observing the rhythm of the cardiac sounds ; and in veins usually continuous, but influenced as to intensity by the sounds of the heart.

Causes of murmurs.   Before the time of Laennec murmurs were not recognized as morbid sounds ; and by him they were attributed to " spasmodic contraction of the heart or arteries."[†] This opinion seems to have been based mainly upon the fancied resemblance which the typical murmur (*bruit de soufflet*) bore to the sound yielded by muscles during contraction.

Corrigan,[‡] as the result of a number of experiments on living cold-blooded animals, and others ingeniously contrived with a view to determine the modifications of sound in membranous conduits under various conditions, arrived at the conclusion that murmur is mainly due to a " current-like motion " of the blood within the heart or vessels.   Thus, according to his doctrine, when the auriculo-ventricular orifice of either side of the heart, or an artery, has suffered absolute narrowing by organic disease, or by pressure ; or relative constriction as the result of simple dilatation of either ventricle, or of aneurism ; the blood in transit through the contracted passage, or into the dilated ventricle or sac, is thrown into a state of intrinsic commotion, or "currents,"

* *Clinical Lectures on the Practice of Medicine*, 1864, p. 472.
† Forbes' translation, p. 559.
‡ *Lancet*, April 4th and 11th, 1829.

whence arises murmur. The transmission of murmur so originating, as well as variations in its intensity, depends mainly upon the degree of tension of the walls in contact with the moving liquid. In the heart unaffected by disease also, a faint murmur must be developed at the auriculo-ventricular orifices, owing to the difference in diameter between these openings and the corresponding ventricles in complete dilatation; "accordingly in the healthiest there is some sound always accompanying its action;" and in dilatation of the ventricles to any extent the sound is loud, and the degree of sound bears a proportion to the size of the ventricle, or to the degree in which this current-like motion exists. In simple dilatation there is a loud, soft, diffused murmuring sound, from which *bruit de soufflet* differs only by its sharpness. He describes the several steps of an experiment, consisting in the transmission of a strong and rapid current of water through a piece of intestine, performed with a view to ascertain precisely the conditions under which *bruit de soufflet* was developed, and its immediate cause; and adds, " whilst the intestine was tense no sound, or a murmur exceedingly indistinct, was heard; but any part being constricted so as to produce an alteration in the motion of the fluid, a very loud *bruit de soufflet* immediately became evident;" the *bruit* was audible, however, only on the distal side of the constriction.

There can be no doubt whatever that the intrinsic collision of the moving liquid, to which alone Sir Dominic Corrigan attributes the occurrence of *bruit,* is a potent, but not the sole cause of this phenomenon. I cannot admit the exclusion of other and no less effective causes, namely, friction and parietal vibration. Moreover, a murmur is actually heard under all circumstances in connexion with liquids moving with a certain degree of rapidity and force through conduits, whether organic or metallic, and quite irrespectively of the tension of their walls. In the leaden pipes laid for the new water-supply of the houses of Dublin from the river Vartry, the water, moving under a pressure of 50 lbs. to the square inch, yields a roaring noise audible at a distance of several yards, and actually stunning to the ear brought into close proximity with it; and in a hose composed of india-rubber, and connected with the water-

pipe for use in my stable, when in action, a murmur is likewise audible, but much less loud and harsh than the former, and rather of a blowing character; differences no doubt due to the better conducting qualities of metal as compared with caoutchouc.

I cannot admit that in a heart the subject of simple dilatation of either ventricle, or of both ventricles, without other organic change, such as narrowing or roughening of the orifice, an afflux-murmur may occur at the auriculo-ventricular opening; and I entirely deny that in a heart free from disease of any kind, a murmur or sound, however faint, is associated with the transit of blood from auricle into ventricle. Such a murmur should be audible at the apex, either coincidently with or immediately succeeding the second sound, when the influx of accumulation takes place; or immediately antecedent to the first sound, when the auricles contract with vigour. It would be, therefore, either a veritable apex-diastolic or post-diastolic, or a presystolic murmur. Of all cardiac murmurs an apex-diastolic is the rarest, that is, a murmur audible at the precordium within the area of apex pulsation only, and strictly coinciding in time with the second cardiac sound. Out of several hundred cases of cardiac disease carefully examined and noted, I can only recall two examples of murmur answering to this description.

Presystolic murmur, until very recently incorrectly designated diastolic,* is *sui generis*, and therefore unmistakeable. This murmur, the special significance of which will be discussed at length further on, is now pretty generally known, and can occur only in connexion with narrowing of either auriculo-ventricular orifice.

Hope† states that " valve-murmurs are occasioned by collision of the particles of the blood against each other, and against the containing solids," and subsequently more at length ‡ " The fact, then, respecting murmurs briefly is, that they may be produced by the vibrations either of the liquid alone, or of the liquid and solids conjointly; and the latter is, without doubt, the more

* *Diseases of the Heart*, by Dr. Hope, 3rd edition, 1839, p. 78.
† *Opus citat*., p. 80.
‡ *Opus citat.*, p. 81, foot note.

frequent case in the heart and arteries, because these solids are elastic.   Hence it is that vibratory tremour is in many instances perceptible to the touch." According to Hope, then, murmur is the result of vibration, whether of the containing parietes, or the contained liquid, or of both; and inferentially, whilst vibration may originate in, and be confined to, the blood, it is, when present in the adjacent solids, always derived from the blood by communication or by friction.   Herein, no doubt, lies the truth in regard to this question.

Inorganic murmurs are, according to Hope, attributable to a threefold cause, viz., attenuation of blood, unfilled arteries, and velocity of current; the first and last mentioned causes giving rise to increased friction, the first and second favouring the occurrence of vibration in the blood, the second supplying the condition for the communication of it to the walls of the vessel, and the third being the immediate active cause by which friction and vibration are developed, and in proportion to its degree, intensified.   Non-organic murmurs, therefore, equally with organic, are the result of vibration and friction.

In experiments on dogs performed by Hope and Marshall Hall, repeated and copious bleedings caused rough systolic cardiac murmur, sharp and strong impulse, and arterial thrill and throbbing.   These phenomena disappeared under two opposite sets of conditions, viz., when the animal was greatly reduced by loss of blood, entailing loss of contractility in the heart; and when the blood was renovated, and thus, at the same time, its volume was increased, and its solid elements proportionately augmented.

When from weakness, murmur, thrill, and throbbing were arrested, they were restored by suspending the animal by the fore legs, manifestly because, owing to the imperfect circulation in the brain in this unnatural posture, the heart and arteries became partially filled, and so, at the same time, the stimulus of imperfect distension was given to the heart, and the conditions for vibration of the blood and of the vessels were again furnished.

Marshall Hall held that vibration was modified, and this murmur caused, by reduction in the quantity or pressure of the

blood, change in the proportion of its elements, and probably in its velocity.[*]

Gendrin[†] holds that murmurs are caused by alteration of structure of the heart or arteries, and changes in the constitution of the blood; and that in the latter case murmur may be heard in all the arteries of the body of a certain calibre.

So-called blood-murmurs are, no doubt, in aggravated cases, audible in the larger arteries of the body generally, but this admission does not imply that such murmurs are regarded as due exclusively to an alteration in the physical constitution of the blood. On the contrary, I believe, and shall endeavour to show in the proper place, that murmurs of this kind are due to nervous reaction upon the walls of the vessels, in no less a degree than to modification in the quantity or in the quality of the blood.

Bouillaud[‡] considers valvular murmur to be nothing more than exaggeration, absolute or relative, of the normal bruit arising from the friction of the blood against the edges of the orifice in its passage through, which is a contributory cause of the first sound of the heart; the other and principal cause of that sound being, according to him, sudden tension of the valves, as previously stated at length (p. 95). If from any cause the force of friction be increased, whether from narrowing of the orifice, or from increased force of ventricular contraction, the friction-element of the first sound is thereby *absolutely* intensified, and becomes murmur.

If, on the other hand, the valve-element of the first sound be partially or entirely suppressed by organic change, then the friction-element is *relatively* exaggerated, or brought prominently forward, and so becomes the cause of murmur. Where the valves are rendered, by organic alteration, incapable of moving, a murmur *replaces* the first sound; but in cases in which the valves move at all, although less freely than in the state of health, the normal sound persists; if there be, however, at the same time, narrowing of the orifice, or a rugged or inadequate state of the valves, *bruit de soufflet, bruit de râpe, or bruit de scie*

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[*] *Gendrin, Leçons sur les Maladies du Cœur*, pp. 14 and 15.

[†] *Opus citat.*

[‡] *Traité Clinique des Maladies du Cœur*, 1835, tom. i.

will *accompany* the first sound. He holds, therefore, that when the valve-element of the first sound is entirely suppressed by structural disorganization, one of the above-mentioned causes of murmur being at the same time present, a murmur of substitution is the result ; but that when any of these causes co-exist with valvular mobility, even though the valves be rough and inadequate to close the orifice, a murmur of accompaniment or a murmur *with* a first sound is heard.

It will be perceived, on reference to Chapters I. and II. (pp. 96 and 115,) in which the sounds of the heart are discussed, that the mere movement of the valves is an element of only secondary value in the causation of them ; sound depending upon the structural integrity or healthy condition of the valves, in virtue of which they are capable of yielding a sound of tension when suddenly put upon the stretch. There may be, therefore, complete suppression of the first sound, notwithstanding that the auriculo-ventricular valves move with freedom, but in such case the valves must be structurally unsound, and the impulse element of the first sound must be masked or abolished.

Da Costa* suggests that in certain cases in which anæmia is a predominant characteristic, murmur may be due to alteration in the tension of the valves, brought about by excited action of the heart, and communicating to the blood vibration which is intensified by the impoverished state of that fluid, but may exist independently of it.

Oppolzer† holds that a thickened and disorganized valve, even though competent to prevent reflux, may give rise to murmur by the altered character of its vibrations, and that even where regurgitation does take place, and is the principal cause of the murmur heard, the abnormal vibration arising from the structural alteration of the valve acts as a reinforcing cause of murmur.

I cannot subscribe the doctrine of tension-murmurs, and I do not believe that valvular murmurs can be fairly attributed to

*American Journal of Medical Sciences, July, 1869 ; and Dublin Quarterly Journal (abstract) November, 1869.

† Dublin Quarterly Journal of Medical Science, November, 1869. Report on Medicine.

this cause, independently of afflux or reflux blood-currents. I have never met with an example of murmur not satisfactorily explicable on other and more rational grounds.

Dr. C. J. B. Williams* is of opinion that murmur is produced by friction of the blood, moved with a certain degree of velocity and force, against irregular surfaces presented by the surrounding solids. The resistance thus given to the current of blood causes sonorous vibrations in the liquid mass, if the latter be urged onwards with sufficient force and rapidity. The resisting medium may be a thickened or irregular valve, a contracted orifice, or a healthy valve opposing resistance through spasmodic contraction of one of the columnæ carneæ. If the velocity be very low there may be no murmur, even with a rigid or partially ossified valve; hence the occasional subsidence of organic murmurs when the circulation has become languid.

Dr. Williams assumes the operation of two other causes of murmur which are, I think, of questionable efficacy; namely, a *relative* narrowing of the orifices of the heart, consequent upon sudden or abrupt contractions of the organ, as in nervous palpitation from excitement, loss of blood, or from inflammation. In such cases a disproportion of the mass of blood to the orifices arises from the suddenness with which it is forced through. I have never heard a murmur of this kind, with the single exception of that following hæmorrhage; but this is due to a cause totally different from that assigned by Dr. Williams, and as belonging to a different class, namely, that of hæmic murmurs, will be discussed separately under that head.†

The other cause of (to me) a doubtful character, to which Williams attributes murmur, is globular dilatation of the ventricle, by which the orifice of exit, though absolutely healthy, is contracted relatively to the ventricle, and the blood in escaping from it, encounters resistance, and is thrown into a state of vibration. I have not met with an example of murmur due to this

* *The Pathology and Diagnosis of Diseases of the Chest,* 3rd edition, 1835, p. 193.

† Skoda (*A Treatise on Auscultation and Percussion,* translated from the fourth edition, by Dr. Markham,) is likewise of this opinion, for he says (p. 211) "A contraction of the heart, more forcible and rapid than ordinary, cannot of itself produce a murmur; and, on the contrary, murmurs (non-organic, *scil.*) may exist when the movements of the heart are slow."

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cause; I have always found slight roughening or crimping of the aortic valve, or atheromatous disease of the vessel immediately above the orifice, in cases where a systolic aortic murmur of indubitably organic nature had existed; and after death an hypertrophed and dilated ventricle was the most notable organic lesion discovered.*

Skoda† concedes the correctness of the generally received opinion, that intra-ventricular murmurs are caused "by friction of the blood against the walls of the heart, or against the valves;" but holds that murmur may be also produced within the cavities of the heart by collision between two streams of blood, one of which is moving in a definite direction, whilst the other is moving in the opposite direction, or in the same direction but more slowly; or by the sudden irruption of a stream into a stationary mass of blood.

Doctor Walshe‡ also advocates the sufficiency of unnatural friction between the blood and the surfaces, and likewise of intrinsic collision between two masses of blood in motion, to occasion murmur. The organic alterations which give rise to abnormal friction are, constriction of the orifices, either simple or with thickening; rigidity; calcification of, and wasting or other growths upon, the valves : and those which occasion collision of blood-streams are, simple insufficiency of the valves to close a widened orifice ; or incomplete valvular occlusion from organic alteration, of whatever kind, of the valves themselves; from shortening of the chordæ tendineæ; atrophy of the columnæ carneæ; adhesion of the segments of the valve to one another, or to the walls of the ventricle or artery : unnatural communication between the different compartments of the heart, or between any of these and one of the arteries, or between the cavities or the arteries and an adventitious chamber. Polypoid growths in the vicinity of the valves, coagula entangled amongst the columnæ carneæ, mere roughness of the endocardium, and abnormal attachments of one of the chordæ tendineæ, whereby

* Dr. Blakiston agrees in opinion with Dr. Williams as to the competency of globular dilatation with hypertrophy of either ventricle, without lesion of the arterial orifice, to give rise to murmur.

† *Opus citat.*, p. 201.

‡ *Diseases of the Heart*, 3rd edition, 1862, p. 86-7.

it is thrown directly across the blood-current, are likewise competent to produce murmur under a strong current of blood.

I have not met with a single example which would warrant me in coinciding with Dr. Walshe in the opinion that, in simple hypertrophy of the left ventricle, without narrowing of the aortic orifice, or valvular lesion, " excess of force of propulsion of naturally constituted blood would seem capable of generating *direct* murmur." Neither am I convinced that palpitation of an hypertrophied heart, with or without disease of the papillary muscles, may, by deranging the action of these muscles, cause mitral regurgitation.[*]

Dr. Stokes very properly urges[†] that, for the production of organic valvular murmur a certain force in the propulsive power of the heart is requisite; and that, in the absence of this essential element in the causation of murmur, the latter not infrequently fails to be heard, even where ossific lesion of the valves exists.

Bamberger[‡] is of opinion that occasionally murmur arises from insufficient tension and after-vibration of the auriculo-ventricular valves, consequent upon fatty degeneration or simple weakening of the papillary muscles.

It will thus be seen that the doctrine of tension-murmurs, but in various forms, has taken firm hold of the minds of some of the ablest pathologists of the present time.

Nevertheless, I cannot subscribe this doctrine. I do not believe that valvular murmurs can arise from modified tension-property of the valves, irrespectively of afflux or reflux blood currents.

Murmur, like normal cardiac sounds, is produced by vibration; thickening and roughness of the valves operate rather by suppressing vibration; and, if they do not give rise to the eddy of obstructed afflux or efflux, or to that of reflux, they cannot be a cause of murmur.

The primary and essential, though remote cause of murmur,

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[*] *Opus citat*, p. 283.

[†] *Diseases of the Heart and Aorta*, 1854, p. 107.

[‡] *Dublin Quarterly Journal of Medical Science*, November, 1869, " Report on Medicine," p. 592.

I hold to be *friction* of the blood-current against the walls or passages where the murmur arises. Friction is a cause of vibration, which is communicated to the walls and to the current, and conducted by the latter in the direction of its flow. Vibration is transmitted by the adjacent solids in accordance with determinate physical laws, being interrupted by dissimilar, and favoured by similar structural constitution of the transmitting medium. Retrograde transmission of vibration, however, through the cardiac or vascular walls, is neutralized by the definite course of the blood-current, whilst progressive or forward transmission through these media is not only favoured, but vibration in them is reinforced, by the blood-stream, itself in a state of vibration from forcible impact at the seat of friction. There is one exception, rather apparent than real, to the non-transmission against the current, of vibratory pulsation of the solids; namely, that which is afforded by regurgitant apex-murmurs arising from reflux at the auriculo-ventricular openings. These murmurs are loudest at the apex, although the current by which they are produced sets in the opposite direction, or towards the base of the heart. But the chordæ tendineæ, owing to their intimate connexion with the mitral and tricuspid valves, their eminently-vibratile properties, and isolated position in the cavities of the ventricles, must receive and communicate to the musculi papillares, and through these to the ventricular walls at the apex, vibrations originating in the valves. The peculiar arrangement of these tendons, moreover, that, namely, by which the greater number of them supplies filaments to two adjacent and opposed valve-segments, renders their *direct* interference with a current of reflux at the auriculo-ventricular orifices unavoidable; they thus, in a measure, create and directly receive the vibrations of that current, and, of necessity, transmit them through the papillary muscles to the apex.

That friction as between a fluid, whether liquid or aeriform, and a solid surface of contact, irrespectively of the regularity of that surface, is capable of yielding vibration audible as murmur and tangible as tremor, is sufficiently illustrated by the passage of water under strong pressure through hose or metallic pipes upon the one hand, and by the phenomena of whistling and

playing upon wind-instruments on the other. In both these cases alike, fluid-friction against the sides of the tube, the lips of the performer, or the mouth-piece of his instrument, becomes a cause of intrinsic molecular commotion, or vibration, in both the fluid and the solid medium ; perceptible in the latter to the sense of touch as *tactile vibration, tremor*, or *fremitus ;* and in both, sensible to the ear as sound more or less harsh, or *murmur.*

Both these phenomena are likewise exemplified in sonorous rhonchus, which, when loud, is always associated with fremitus.

Murmur and fremitus, though due to a common cause, are not always associated, because fremitus requires for its production stronger and coarser vibration than does murmur.

Hence fremitus is a much less constant and invariable phenomenon than murmur ; and hence likewise it is, that whilst both are frequently associated, whenever that association is dissolved by disappearance of either phenomenon, fremitus is that which ceases to be manifested.

Within the last ten years much has been done experimentally, and written, especially in France, with the object of elucidating the phenomena of cardiac and respiratory murmurs. The results obtained have tended, in a singular manner, to confirm those of Sir Dominic Corrigan already referred to ; and to establish the general correctness of the views as to the cause of murmur, and the laws of its propagation, expressed by that eminent physician so long ago as 1829.

The writers to whom I especially refer are M.M. Chauveau,[*] Boudet,[†] and L. Bergeon.[‡] The vascular bruit has been shown, in the opinion of these writers, to be due to an intrinsic commotion in the circulating fluid, designated *" veine fluide,"* by Savart. M. Bergeon says : " Whenever a liquid flows from a reservoir,

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[*] " Etudes Pratiques sur les Murmurs Vasculaires, ou Bruits de Souffle, et sur leur valeur Séméiologique," *Gazette Médicale de Paris*, 1858, p. 247. For this and the subsequent references, as well as for an able summary of this subject generally, I am indebted to the writer of Article ii., " On the Physical Theory of Murmurs, vascular, cardiac, and respiratory," in the *British and Foreign Medico-Chirurgical Review* for July 1873.

[†] " Recherches Physiologique sur le Méchanisme des Bruits Respiratoires," *Gazette Hebdomadaire*, 1863, p. 789.

[‡] *Des Causes et du Mechanisme du Bruit de Soufflet*, Paris, 1868 ; and *Théorie des Bruits Physiologiques de la Respiration*, Paris, 1869.

through a vertical or horizontal orifice, the stream takes the form of a jet, to which Savart gave the name of ' fluid vein.' "

"The ' fluid vein ' is essentially composed of two parts, the first calm, transparent, like a stem of crystal; the second agitated, without transparency, but so far possessed of regular form, that it can be seen to be divided into a certain number of elongated swellings (of which the maximum diameter is always larger than that of the orifice), separated by narrower portions. This nodal appearance of itself shows that the liquid vibrates, and by means of the electric light Savart showed that the flow is not really continuous. The ' fluid vein' in part consists of separate drops of the fluid, succeeding one another at regular intervals, each drop changing its form in passing from point to point."

The production of a "fluid vein," or vibrating axial current, whether in a liquid or a gas, depends essentially upon its transmission with a certain force through a narrow orifice into a larger space beyond, whether that space contain air or liquid. And in this relatively larger or (in regard to the direction of the current) distal space alone the "fluid vein " or sonorous vibration is generated. This, as shown by Bergeon, and as long before correctly stated by Corrigan, is due to the different pressure experienced by the fluid on opposite sides of the constricted orifice To the same conclusion leads an experiment performed by Chauveau on a pithed horse, artificial respiration having been kept up; from this and other experiments he deduces the following general law :

" Bruit de souffle is always produced by the vibrations of an intra-vascular *veine fluide*, and such a *veine* is constantly formed whenever the blood passes with a certain force from a narrowed into an actually or relatively dilated part of the circulatory system." But manifestly, under this law, something besides intra-vascular narrowing may produce a " fluid vein," and something other than " fluid vein " may produce *bruit de souffle*.

Roughness of the intra-cardial and intra-vascular surface as a cause of murmur, has been summarily set aside by Chauveau, but as I think, on insufficient evidence,

On the same principle, that of a " fluid vein," Chauveau like-

wise explains anæmic murmurs at the base of the heart, and in the great veins of the neck.

In anæmia there is a general reduction in the volume of the blood; the chambers and orifices of the heart, and the blood vessels generally, with two exceptions, accommodate their dia meter to the reduced volume of blood. The exceptions are: (*a*) the aorta and pulmonary artery, which, owing to the absence of the contractile and the preponderance of the elastic element in their walls, cannot reduce their diameter proportionately to that of the current passing through them. Hence a "fluid vein" and murmur of exit. (*b*) The roots of the innominate veins are fixed and kept permanently dilated by the cervical fascia, which not only ensheaths them, but is connected with the sternum, clavicle, and first rib Hence, whilst the jugular and sub-clavian veins above accommodate themselves to the reduced diameter of their respective currents, the commencing portion of the innominate veins, being incapable of a reduction of calibre, give origin to a "fluid vein," and a venous hum, or *bruit de diable*.

This is a most ingenious and apparently a satisfactory ex-planation of venous murmur. Against Chauveau's theory, as applied to the heart, the apparently insuperable objection may be urged, that it necessarily implies a murmur of diastole at the auriculo-ventricular orifices in the normal state of the heart Corrigan anticipated this objection, by alleging that normally such a murmur is actually but faintly audible at the points mentioned. But, in point of fact, no such murmur can be detected in the physiological state.*

Murmurs are governed by the laws of ordinary sound in re-gard to their pitch, quality, and intensity.

The *pitch* or key of a murmur is determined by the number of sonorous undulations occurring within a given time; the greater the number of such, the higher the pitch; and conversely. Some murmurs are of so low a pitch that they scarcely form a con-tinuous sound, whilst others, on the contrary, have a pitch so high that they are musical in character. As a rule, to which I

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* Further, the remarkable difference in the *quality* of murmurs would not be ex plicable on this theory.

am not acquainted with a single valid exception, very high pitched, and hoarse or low pitched murmurs, are of organic or of thromboid origin, and therefore of less favourable significance than those of an intermediate key.

The *quality* of a murmur, expressed by its harshness or the opposite, depends upon the abruptness of the vibrations to which it is due. Harsh grating murmurs are the result of the attrition of two uneven and dense surfaces against one another, or of a strong current upon solid, rugged, and projecting points of surface. Soft murmurs are caused by the movement of currents over smooth and even surfaces; and therefore, whilst they may indicate formidable valvular disorganization, they cannot be regarded as evidence of atheromatous or calcareous transformation.

I cannot agree with Hope in holding that harshness depends upon the configuration of the orifice, rather than upon the character of the disorganizing medium. Nor can I admit with him, that " osseous disease " of the cardiac valves or vascular walls, if covered by the lining membrane, may give rise to bellows murmur; and that, on the contrary, fibrous and fibro-cartilaginous structures may give origin to rasping and sawing murmurs.

The intensity of a murmur is directly as the strength of the primary impulse, the proximity of the seat of origin to the ear, and the conducting properties of the intermediate structures. Proximity influences the loudness or distinctness of a murmur; but it certainly is an error to assert, as Hope has done, that it modifies, much less determines, the pitch of a murmur. It has been shown experimentally by Biot, that distance has no influence on pitch. If this were not so, musical notes of different pitches, heard at a distance, would run into one another and be out of harmony; but such is notoriously not the case. Dr. Walshe, writing on this subject says,* " The mere distance of the site of production of murmurs from the surface, can, in theory, have no direct influence on their pitch. The same physical and dynamical conditions will generate sounds of the same pitch, whether they be close to, or as far as possible from, the chest-wall."

Murmurs have been classified according to their position, their

* *Opus citat.*, p. 90.

rhythm, relationship to the normal sounds, pitch, and quality; and a complete definition or description of a murmur must include a reference to each of these characteristics. This is a matter of some moment in regard to intelligible description; and it were much to be desired that this, or some other equally convenient formula for the designation of cardiac murmurs, should be universally adopted by physicians. Till a universally recognized standard of this kind be established, laxity of description, and consequent confusion and misinterpretation will continue to be, as at present, unavoidable.

The order in which I have just mentioned the several characteristic features of a murmur is the analytical. The synthetical method of description, however, will be found more convenient as applied to any particular murmur, as is practically found to be the case in regard to other matters, in specifying one of a multitude of objects of the same kind. I shall revert to this subject after having treated of the different characteristics of murmurs as above sketched.

The site or *point of maximum intensity* of a murmur is of great importance in regard to differential diagnosis. Murmurs have been divided according to their site into *apex murmurs*, and *basic murmurs;* and each of these groups has been subdivided into left and right. Thus there are, in the order of their frequency, *left* and *right* apex murmurs, and *right and left* basic murmurs.

The determination of this point of maximum intensity of a murmur is a purely acoustic operation, in the performance of which, however, great readiness and precision are acquired by practice and close attention.

The *rhythm* of a murmur has been interpreted by the majority of writers on diseases of the heart, as having reference only to those periods of the cardiac cycle which are covered by the normal sounds, and is by medical men generally so regarded. Nevertheless, it is an unwarranted limitation of the meaning of the term, because it implies that during the intervals between the sounds, or the short and the long pause respectively, murmur is never heard.

That murmur is, however, occasionally and not unfrequently

audible during the normal periods of silence, there can be no longer any doubt; and furthermore, that murmurs of this particular rhythm have, in regard to each of the two pauses, a specific significance of great diagnostic value, I am quite convinced, and undertake in the sequel to shew.

Gendrin* seems to have been the first to recognize this important distinction, to which, indeed, his subdivision of the normal periods of silence (page 82) led him by logical necessity.

Gendrin's classification of murmurs on this basis is, however, not only eminently unpractical, but highly erroneous, as will appear further on; but to him, nevertheless, belongs the merit of having been the first to recognize the existence of intersonal murmurs, and to introduce corresponding designations, even though somewhat overdrawn, and in many instances incorrectly applied.

Doctor Walshe not only recognizes the existence of murmurs falling within the periods of cardiac silence, as distinguished from those of sound, but proposes a much more euphonious nomenclature. He says, " But they [murmurs] are not necessarily synchronous with either systole or diastole. They may be pre-systolic, systolic, or post-systolic ; pre-diastolic, diastolic, or post-diastolic."†

Many years ago, before I had the pleasure and advantage of reading Dr. Walshe's valuable work, I had, from clinical study, perceived the necessity of admitting intersonal murmurs, and actually used the designations I am happy to find Dr. Walshe has anticipated me in proposing. The appreciation, by two independent observers, even though at different periods, of a special and distinctive morbid phenomenon, and referred by them to the same cause, is, *pro tanto*, evidence of the accuracy of their respective observations.

At the meeting of the British Medical Association at Oxford in 1868, I read a paper in which I set forth my views on this subject, and gave cases in illustration. The subjoined diagram will be found to render this classification simple and intelligible :

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* *Leçons sur les Maladies du Cœur,* 1841-42.

† *A Practical Treatise on the Diseases of the Heart and Great Vessels,* 3rd edition, 1862, p. 85.
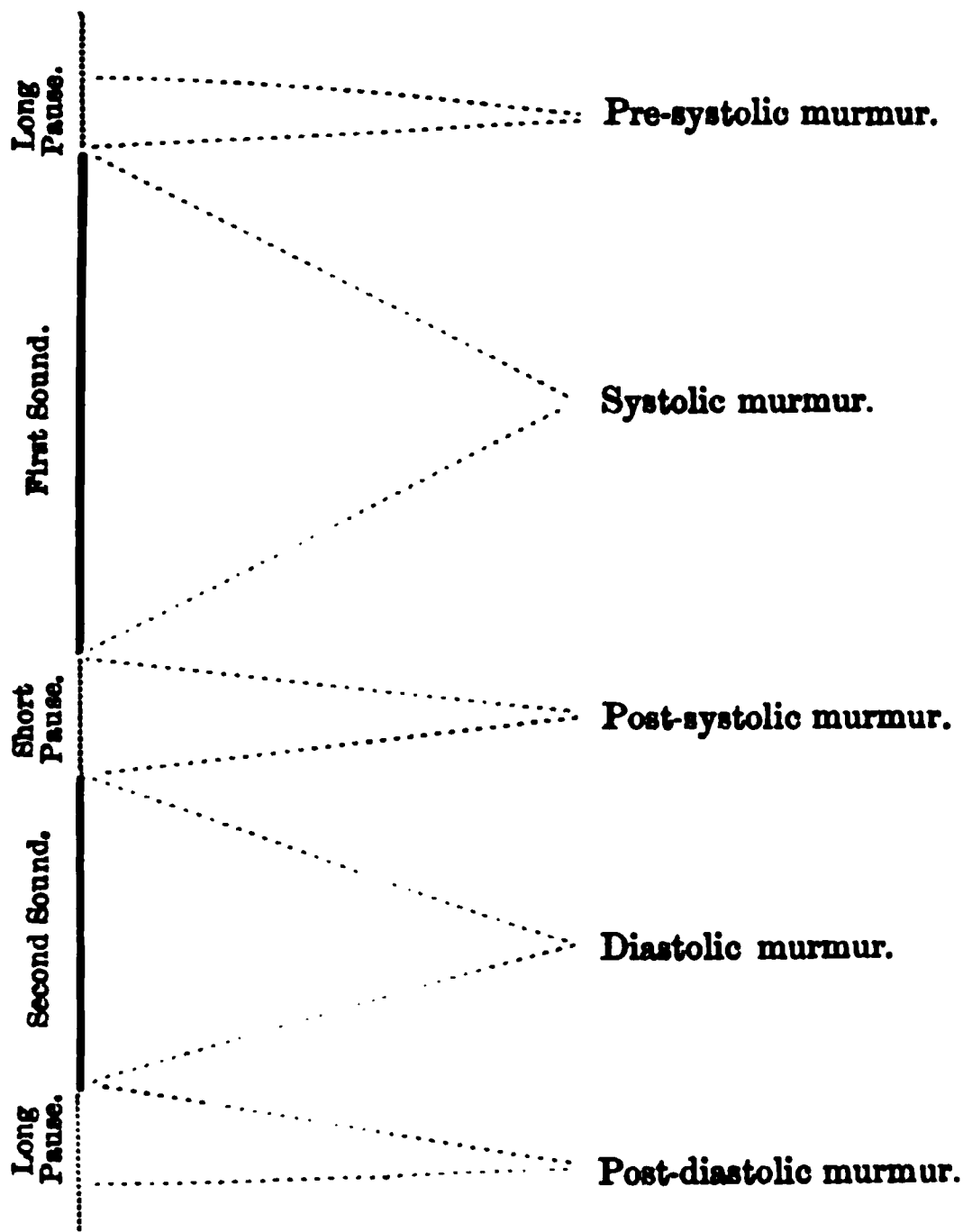
The preceding classification of cardiac murmurs may seem to involve unnecessary refinement; that is, to imply distinctions not actually to be met with in disease. I am aware that many highly respectable pathologists, and a few of special authority on this subject, hold that if it be not quite impossible in practice to draw the distinction between *sonal* and *intersonal* murmurs as above indicated, at least no information of value, in regard to diagnosis or treatment, can be deduced from it. To this objection, to which I pay all due respect because of its authorship, I shall give a very simple answer. If the distinction be founded in nature, and murmurs of the special rhythm of those named intersonal be of actual, even though rare occurrence in disease, then it is sufficiently warranted, and needs no further justification, quite irrespectively of its imputed value as an aid to differential diagnosis.

Of the existence and special diagnostic value of at least one, and that the typical form of this class of murmurs, namely, the presystolic, doubt is no longer entertained by anybody not altogether impenetrable to the light of progress. Yet less than thirty years ago the existence of a murmur of this special rhythm was utterly unknown, and notwithstanding the suggestive intimation of its distinct identity given by Walshe in England, and the more positive affirmation of Markham and Gairdner to the same effect, presystolic murmur had not a place assigned to it in the established semeiology of English medical literature till 1867, when Doctor Peacock fully admitted its separate entity and special significance,* and attributed his conversion to Dr. Gairdner's writings and mine on this subject.

Bouillaud in 1835† regarded the murmur of auriculo-ventricular constriction as diastolic in rhythm, as may be inferred from a passage in his *prolégomène*, in which, whilst discussing multiplication of the sounds of the heart, he adduces an example of quadruple sounds ; the first associated with slight murmur, the second and third sharp and harsh, and the fourth represented by a bellows murmur. The second and third he regarded as a reduplicated second sound, " as if the ventricle had not the power to fill itself, save in two movements, or in two efforts." Herein Bouillaud implies, what he elsewhere, as already shown, distinctly states, that in his opinion, the second sound is caused by the passage of blood into the ventricles ; and further, that the murmur of auriculo-ventricular constriction, if such exist, must be diastolic in rhythm, or one accompanying or replacing the second sound of the heart.

Gendrin in 1841 ‡ subdivided the periods of cardiac silence, as previously stated at length, into the *péridiastole*, and *présystole*, the *périsystole* and *prédiastole* respectively ; but that he misapprehended the precise rhythm of at least one, and that the most important of the murmurs which should correspond to these sub-divisions, will be manifest from the following references to his work. He says : " When the column of blood, in

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* *British and Foreign Medico-Chirurgical Review*, No. lxxx., October, 1867.

† *Opus citat.*

‡ *Leçons sur les Maladies du Cœur.*

passing from the auricles into the ventricles, encounters rugosities at the auriculo-ventricular orifices, we hear a *prédiastolic* murmur. If the rugosities extend to the free edge of the valves, this murmur is prolonged to the second sound, and ends with it. If the alteration occupy exclusively the free edge of the valves, without engaging their attached border, or the auriculo-ventri- cular orifice properly so called, the murmur *commences only with the diastole, and terminates with it*; it is then *diastolic*. When the murmur is *prédiastolic* we are warranted in suspecting that narrowing of one of the auriculo-ventricular orifices exists. We may positively affirm that such is the case, if there exist at the same time doubling of the second sound (*percussion diastolique*). The blood occupies a longer time in traversing the narrowed auriculo-ventricular orifice, and the murmur is the more prolonged, the greater the contraction of the orifice is."

"*Prédiastolic* murmurs, which are produced by obstacles to the free and ready passage of the blood through the auriculo-ventricular openings, are heard with greatest distinctness at the middle of the vertical diameter of the precordial region; they are lost at the base, and towards the apex of the heart."

"Systolic, périsystolic, and prédiastolic murmurs of endocardial origin are often combined. Thus, if rugosities exist on both surfaces of the mitral valve, and are very prominent, a *prédias- tolic* murmur may be heard coincidently with the passage of blood through the auriculo-ventricular orifice, and another, a systolic murmur, produced when the wave of blood strikes against the surface of the valve during ventricular contraction ; these two murmurs are likewise met with when the mitral valve is inadequate."

He also writes :[*] " Auriculo-ventricular narrowings most usu- ally have their seat at the summit of the valves, then co-herent ; thence results a cone-shaped infundibulum, at the bottom of which is found the contracted orifice, forming a sort of muzzle with two or three lips. If, in the bottom of this infundibulum with thick and indurated walls, inequalities, or small warty and fig-like tumors be found, *the diastole is preceded and accom- panied* by a vibratory thrill which is perceived in greatest in-

[*] *Opus citat.*, p. 130.

tensity at the middle of the vertical diameter of the heart, on its left border, if the lesion be, as most commonly it is, at the left *auriculo-ventricular orifice.*

" The *prédiastolic* vibratile thrill, which is produced in such cases, is always feeble, at least as regards the mechanical transmission of the vibrations to the walls of the chest.

" We often meet with the valvular lesion just indicated, as a cause of *prédiastolic* vibratile thrill, to coincide with different lesions, likewise previously described, and to the presence of which is to be attributed systolic vibratory thrill. In this case there are two thrills separated by a period of silence, *which measures the length of the interval between the diastole and the systole.*"*

The extracts above given from Gendrin's book show conclusively that he has no claim to the merit of having recognized the diagnostic significance of presystolic murmur, because he has quite mistaken for it a murmur of different origin and rhythm, namely, one associated with the second sound of the heart, and therefore separated in time from the presystolic murmur by the greater portion of the long pause. Nor has Hope any better claim to the distinction of having recognized, much less discovered, the presystolic murmur, as will appear from the following extracts from his great work.

He says :† " Will it be said that the auricular contraction, previous to the ventricular, should create a murmur? I have looked for it carefully, and have only once been able to suspect it, without being able to assure myself of its existence. Theoretical reasoning seems to countenance this result of observation; for, as the auricular systole is slight, the quantity of blood injected by it is not considerable, and as the ventricle is already *full*, it cannot admit that extra quantity necessary to bring it to the state of distension, without offering a resistance to its ingress, which must greatly retard the force and velocity of the current; a force, indeed, which can never be great, because the auricles are not only weak muscles, but are unsupported by valves behind."

* *Opus citat.*, p. 111. The italics, I need scarcely observe, are not in the original.
† *A Treatise on the Diseases of the Heart and Great Vessels,* 3rd edition, 1839, p. 79.

Discussing (p. 80) diastolic murmur at the tricuspid orifice, he says: "It is so rare that (abstracting *pulmonic and aortic regurgitant murmurs, with which it is apt to be confounded*) I am not satisfied that I have ever met with an instance of it." "The reason assigned for the rarity of *diastolic* murmurs in the contracted mitral valve, namely, the feebleness of the current of blood, applies equally to the tricuspid."

He describes (p. 78) murmur of mitral contraction as rare, "*diastolic*," and liable to be confounded with that of aortic reflux, from which, he says, it is to be distinguished only by its being confined to the apex.

Finally, in reference to the differential diagnosis of aortic regurgitation, he writes: "The murmur accompanying the *second sound* I am inclined to attribute, perhaps entirely, to the same (viz., inflammatory) constriction affecting the *auriculo-ventricular valves*, or, I should have added, occasioning *patency of the sigmoid valves* ;" and again (p. 74-5) "It may be diagnosed from mitral *diastolic*, always feeble, but inaudible at the aortic valve, *as they both occur during the diastole*, by its being loudest at the aortic orifice, though prolonged towards the apex."[*]

Thus Hope states, *passim*, that the murmur of mitral obstruction is diastolic in time, and that by its rhythm it is liable to be confounded with aortic and pulmonic reflux murmurs. Hence it is clear he failed to identify the presystolic murmur of mitral constriction, which, in its ordinary form, occupying not the initial but the terminal portion of the long or diastolic pause, is in no manner associated with the diastolic or second sound of the heart; and therefore, not in the remotest degree liable to be confounded with the regurgitant murmur of aortic or pulmonic inadequacy.

A *veritable* apex diastolic murmur is undoubtedly "rare," as Hope describes it. I have met with only two examples of such murmur, and Doctor Stokes informs me he has met with another. In one of my cases I had the advantage of an autopsy, the particulars of which I shall give in connexion with a more lengthened discussion of this subject in a future page.

[*] The italics in the preceding quotations, with the exception of the word "*full*," are not in the original.

Skoda, discussing the physical diagnosis of mitral constriction, states as follows :* " *In the left ventricle, during its diastole, a sound unaccompanied by a murmur* (second sound) indicates that there is no constriction of the left auriculo-ventricular opening, and that the blood, in passing from the left auricle into the left ventricle, does not flow over any roughened surface.

" *A murmur accompanied by a sound, or a murmur alone,* indicates either constriction of the mitral orifice, with roughness of the narrowed surface of the canal, or the presence of rough eminences upon the auricular surface of the mitral valves, unaccompanied by constriction of the opening."

" If the left auriculo-ventricular opening be constricted (and this condition is almost always associated with defect of the mitral valves), the *diastolic murmur* is generally loud and prolonged, the systolic being weak and of short duration," etc.

Thus he lays it down that a second sound in the left ventricle, unconnected with murmur, is conclusive evidence against mitral constriction or roughness of surface ; and conversely, that a murmur accompanying the second sound, or superseding it, is positive evidence of mitral narrowing; and finally, if the mitral orifice be narrowed, as distinguished from simple roughness of surface, the murmur with, or replacing the second sound, is loud and prolonged.

Doctor Stokes says :† "In the ordinary cases of mitral murmur we cannot say whether the murmur is " constrictive " or " regurgitant," or constrictive and regurgitant ; and we must reject a large proportion of descriptions of phenomena which, although the changes they are supposed to indicate be familiar to anatomists, are themselves of doubtful value."

Dr. Bellingham states :‡ "Disease of the mitral valve or orifice, causing an impediment to the current from the left auricle into the left ventricle, is sufficiently common ; but a *diastolic* murmur from this cause is excessively rare ; so rare that the most excellent observers have never heard it. Indeed, the current from

* *A Treatise on Auscultation and Percussion ;* Markham's translation, 1853, pp. 232-3-4.

† *Diseases of the Heart and Aorta,* 1854, p. 181.

‡ *On Diseases of the Heart,* 1857, part ii., p. 383-4.

the auricle into the ventricle is generally too feeble to develope one, and this condition of the mitral valve and orifice is characterized rather by a diminution in the intensity of the *second sound* than by a murmur.

" I cannot help thinking that, in the cases in which a murmur from this cause is said to have been heard, the murmur of mitral regurgitation was mistaken for it, which sometimes, instead of accompanying the ventricular systole and the first sound, *follows* it, and thus obscures the second sound of the heart."

No doubt, a diastolic murmur from this cause is excessively rare. I also freely admit that the current of blood from the auricle into the ventricle is too feeble under ordinary circumstances to develope a murmur, save towards the termination of auricular systole, or during its *momentum*, when a murmur not only may occur in mitral contraction, but as a matter of fact is very rarely absent in cases of that lesion.

Occasionally an example is met with of murmur immediately following the first sound, or postsystolic murmur; but such a murmur belongs to, and is distinctive of, mitral regurgitation, not mitral contraction, which may or may not be likewise present; it extends into the short or systolic pause, and may even be so far prolonged, though it rarely is so, as to touch the second sound, in which case alone it could be regarded as in any sense diastolic. But whether it be or be not so prolonged as to give rise to a mistake in regard to its proper rhythm, under no conceivable circumstances can it be confounded with the murmur of mitral obstruction or constriction, which is *never* exclusively diastolic, in the sense of being synchronous with the second sound; *never* engages any portion of the short or systolic pause; *always* precedes the first sound; and *always* engages the long or diastolic pause.

Dr. Ormerod, discussing some novel forms of murmur,[*] writes: " A murmur, audible at or near the apex only, accompanying *the first sound*, rising in intensity with the continuance of the first sound, and ending suddenly with a snap at its closure, admits of more than one interpretation. Its seat is unquestioned : the mitral or the tricuspid orifice. But is it due to *obstruction* to

* *British Medical Journal*, August 6th, 1864.

the flow of blood from the auricle to the ventricle during the auricular systole, or to imperfection of the auriculo-ventricular valve, allowing regurgitation during the contraction of the ventricle ? To the first explanation we may object, that a direct mitral murmur, such as is generally admitted to be of this nature, is quite unlike the murmur under consideration; it is soft, and closely resembles the murmurs of sigmoid regurgitation. Such a murmur is very rare; while the murmur under consideration is not at all uncommon. It may be questioned, too, whether the contraction of the auricles, slight and momentary as experimentalists report it to be, is capable of producing so loud a sound. To the second explanation I do not see that there is any insuperable objection."

It is manifest from this extract that Dr. Ormerod, so late as 1864, regarded the rhythm of direct or mitral obstructive murmur as systolic, or "accompanying the first sound;" and from the objections which he urges against this hypothetical view, and which have reference to the quality and the rarity, not to the rhythm of the murmur, it is equally clear that he was unacquainted with the distinctive characteristics of presystolic murmur. The quality (softness) which he assigns to it is, moreover, not that which really belongs to it.

Dr. Blakiston says:* "The chief physical sign of mitral obstruction is a *diastolic* murmur; it is rarely, however, that such a murmur is engendered, because the size of the auriculo-ventricular opening is so large," etc. Here the rhythm of the murmur is likewise mistaken as being "diastolic," an error which, by fixing attention exclusively upon the second sound, may lead either to non-observance of the murmur where it actually exists, or to non-recognition of its true nature even when detected.

Doctor Walshe states :† "A diastolic murmur of maximum force, *immediately above and about the left apex*, and conducted in the same direction, though less extensively, as systolic murmur of the same seat, indicates *obstructive* narrowing of the mitral orifice, or simple roughness of the auricular surface of the mitral valve ; or both states combined.

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* *Diseases of the Heart*, 1865, p. 246.
† *Diseases of the Heart*, third edition, 1862, p. 103-4.

" This murmur is commonly spoken of as diastolic in rhythm; but, in point of fact, it is rather postdiastolic, or *præsystolic*, than precisely coincident with the diastole."

Further on he adds* : " I have already stated that the rhythm of this murmur is rather postdiastolic, or præsystolic, than actually diastolic."

It appears from these extracts that Dr. Walshe had a not very definite, but still, *pro tanto*, a correct appreciation of the rhythm of this murmur, and that he was, beyond all doubt, the first in this country to point it out.

Sir William Gull fully admitted† the diagnostic value of presystolic murmur. Yet three years later I find Dr. Tanner stating as follows :‡

" A diastolic murmur most distinct from the centre of the sternum (on a level with the third intercostal space) upwards towards the base, with a jerking pulse, is indicative of aortic regurgitation ; while a *diastolic murmur*, most distinct from the fourth left intercostal space, onwards towards the apex, with an irregular small pulse, is the result of *mitral obstruction*." And Dr. Niemeyer in his celebrated work§ lays down rules for the identification of this murmur as follows, viz. : " Upon auscultation we almost always hear a long-drawn murmur at the apex during diastole." He adds, " In addition, we can, of course, hear the *second sound* propagated from the arteries, unless the murmur be too loud."

Manifestly Dr. Tanner failed even to identify the murmur to which so much significance attaches; for he describes it as diastolic in time, and also as being associated with an irregular small pulse, a symptom which, though met with in connexion with mitral stenosis in its last stage, or when death is imminent, most certainly does not belong to that lesion at a time when differential diagnosis is of any consequence to the patient.

Niemeyer, whilst associating with this murmur " presystolic purring," strangely and inconsistently describes it as " diastolic," and associates it with the " second sound."

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* *Opus citat.*, p. 372.
† *Medical Times and Gazette*, January 27th, 1866.
‡ *The Practice of Medicine*, sixth edition, 1869, vol. i., p. 619.
§ *A Text Book of Practical Medicine*, second edition, 1869, vol. i., p. 356.

Dr. Aitken says :* " The murmur indicative of obstructive narrowing of the mitral valve is a *ventricular diastolic murmur* heard in maximum force *immediately above and about the left apex.*"

The late eminent Professor Trousseau likewise described this murmur as *diastolic* in time, as shown by a passage in his recently published lecture on " Organic Diseases of the Heart."†

To M. Fauvel belongs the merit of having been the first to identify the presystolic murmur, and fully appreciate its pathological significance, as will appear from the following quotations from his memoir published in 1843.‡ He gives Beau the credit of having been the first to expose the fallacy of attributing a diastolic rhythm to the murmur of auriculo-ventricular constriction, by declaring that " There is not on record a single well-authenticated example of diastolic murmur at the apex in connexion with narrowing of the auriculo-ventricular orifice."

In 1843, Fauvel observed in the person of a discharged soldier an intense bruit de râpe preceding the first sound, ending with it, and loudest at the apex and to the left. Four new cases followed, and of these three were fatal.

Case 1, a female, aged fifty, the subject of a chronic cerebral affection ; strong impulse and extended precordial dullness. A strong bruit de râpe loudest at the level of the fifth rib, to the left of the nipple, and growing faint towards the right ; commencing in the long pause, and ending at the instant of the first sound ; pulsation intermittent ; pulse small and irregular, and no œdema. Death from the cerebral affection. The left auriculo-ventricular orifice was contracted to the size of the tip of the middle finger, and cartilaginous and warty on the auricular aspect ; slight hypertrophy of the left ventricle, but no dilatation. In reference to this case he remarks : " It was of importance in this, that it exhibited a considerable narrowing of the auriculo-ventricular orifice, coinciding with an abnormal bruit associated with the first sound in such a way that it commenced almost in

* *The Science and Practice of Medicine,* second edition, vol. ii., p. 717.

† *Lectures on Clinical Medicine,* New Sydenham Society, vol. iii., 1870, p. 413.

‡ "Mémoire sur les signes stethoscopiques du Rétrécissement de l'Orifice Auriculo-ventriculaire gauche du Cœur," *Archives Générales de Médecine,* tom. i., 1843.

the middle of the long pause, and ended at the instant when the first sound was heard."

Case 2 exhibited a rough murmur commencing before the normal first sound and ending with it, so as to impart to it a very hoarse pitch (fortiment enroué). On examination after death the mitral was found contracted to the size of the little finger.

Case 3 yielded a bruit-de râpe preceding and covering the normal first sound at the apex and to the left. There were likewise an apex and an aortic postsystolic murmur. The mitral orifice was found contracted to the size of the little finger, and the valves inadequate; the aortic orifice was contracted, and rough by excrescences. He concludes, correctly, that the presystolic murmur at the apex was due to mitral obstruction, and the postsystolic murmur to mitral reflux. The three last-mentioned patients were females.

Case 4, a man aged thirty-two. "A bruit de râpe, which commenced in the long pause an instant after the second sound, and ended with the first." Left hospital improved.

Case 5, a man of fifty. "An abnormal bruit at the apex and to left, commencing a little before the first sound, and imparting to it a hoarse character. There was likewise the murmur of aortic reflux. On *post mortem* examination the mitral orifice was found barely to admit the index finger, and the aortic valves were rough and inadequate." He adds: "From all this it follows, as the first consequence, that a bruit de. râpe localized at the apex and to the left, and immediately preceding the normal first sound, may be the only morbid sound corresponding to a very considerable narrowing of the left auriculoventricular orifice, without inadequacy."

The accuracy with which the premises are above stated, and the justness of the conclusion drawn from them, leave nothing to be added. He designates the murmur as "presystolic," a term for which he is indebted to Gendrin. But as already stated, Gendrin used it with a different meaning. He says " It is the most probable sign of narrowing of the mitral orifice, but I don't pronounce it a certain sign, because the small number of facts on which the conclusion is based does not admit of its being

otherwise regarded than provisional, and as requiring the sanction of fresh observations." In reference to this very modest declaration it may be said that the confirmation intimated as necessary has been since amply supplied,

M. Hérard, ten years later, questioned two of Fauvel's observations, because, forsooth, the murmur is described "as commencing soon after the second sound." He casts doubt upon the entire doctrine of his distinguished compatriot, and strenuously labours, no doubt in good faith, to bring back the state of chaos which he had dissipated. Thus, he maintains that mitral narrowing may give rise to a *systolic*, a *presystolic*, or a *diastolic* murmur, and that in old subjects it may be unrepresented by murmur of any kind.*

Hérard gives a list of cases in which an opportunity for correcting the diagnosis by dissection was afforded, and another in which the diagnosis remained uncertain. The former I shall give in summary.

Case 1, a female aged twenty-four; a rough systolic murmur synchronous with the carotid pulse. On *post mortem* examination the mitral orifice was found reduced to the size of the little finger, with smooth and thick edges, but the valve was competent as proved by the water test. All other openings and valves healthy. The circumstances above mentioned warrant the inquiry whether the rhythm of the murmur was not misapprehended ? It must have been presystolic, however masked.

Case 2, a man aged thirty-seven. Double apex murmur (systolic and diastolic) and fremissement. The mitral orifice was found reduced to the size of the little finger, funnel-shaped and rigid.

"Diastolic" murmur in this case was, I suspect, really postdiastolic, and may be explained by the extreme narrowing which existed; owing to which the *passive* disgorgement of the left auricle at the commencement of ventricular diastole, and therefore synchronous with the second sound, may have given rise to murmur extending beyond it. When the auricle had

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* *Archives Générales de Médecine*, tom. ii., 1853. For this and many of the succeeding references I am indebted to Dr. Hilton Fagge, whose valuable memoir in the *Guys' Hospital Reports*, third series, vol. xvi., is indeed a repertory on this subject.

partially unloaded itself murmur ceased, to return with the systole of the auricle at the end of the long pause.

Case 3, a man aged thirty-five. Systolic murmur at base, and double murmur at apex. The aortic valves were found thickened, and the aorta rough by atheromatous plates. The mitral orifice was contracted, and one segment of the mitral valve was rigid and held open by an ossified papillary muscle and tendinous chord.

Case 4, a man. Diastolic murmur at the apex. Fibro-cartilaginous transformation and narrowing of the mitral opening were found.

Case 5, a woman aged twenty-two. Double apex murmur (systolic and diastolic) and *fremitus felinus*. The mitral opening was found narrowed to the size of the little finger, rigid, and funnel-shaped. The tricuspid orifice was in the early stage of similar change. The observations made in reference to Case 2 may be likewise applied to this.

In the discussion of Hérard's memoir M. Beau reiterated his peculiar doctrine as to the rhythm of the different movements of the heart, maintaining that ventricular diastole occurs in " le premier temps," and no portion of it in " le second temps," save in the case of inadequacy of the aortic valve, when it is of necessity prolonged into the period of the second sound. He affirmed, moreover, that diastolic murmur at the apex is always due to aortic reflux, and *never* to mitral narrowing.*

The entire of this doctrine is now very properly regarded as heretical, the justly high authority of M. Beau notwithstanding.

In 1859 M. Raclet† doubted the possibility of identifying the peculiar rhythm of presystolic murmur in practice, and inclined to regard it as systolic, and in 1862 Durozier sneered at it as a myth.‡

Three years anterior to the last-mentioned date, however, Dr. Austin Flint of Philadelphia had in effect admitted the identity and distinctive character of presystolic murmur;§ he designates

* *Archives Générales de Médecine*, 1854.

† *Traité de Diagnostic Médical*, 2ème ed. 1859, p. 290.

‡ *Archives Gén.* ser. v., tom. xx.

§ *Pract. Treatise on the Diagnosis, Pathology. and Treatment of Diseases of the Heart*, Philadelphia, 1859.

it as diastolic, but adds that it is more strictly presystolic. A few years later he affirms, in more positive terms, its distinctive rhythm, and more accurately describes its quality.*

" Mitral direct murmur " he says " precedes the first sound. The mitral direct current of blood, therefore, occurs just before the ventricular systole ; it continues up to the ventricular systole, and must of course cease when the ventricles contract. The contraction of the ventricles causing the first sound of the heart, it follows that the mitral direct current caused by the auricular contraction must take place just before the first sound, that it must continue to the first sound, and that it cannot continue an instant after the first sound.

" In my work on *Diseases of the Heart* I have said that this murmur (presystolic) is generally soft. My experience since that work was written has shown me that this statement is incorrect.

" I should say that whilst the mitral systolic murmur is much more frequent in its occurrence than the mitral direct, the former, indeed, being the most common of all the murmurs, the mitral direct is observed quite as often without as with the mitral systolic. Between the mitral direct and the mitral systolic (when they concur) occur the apex beat, first sound, and carotid pulse. (? ?)

" The murmur, however, may be produced by the flowing of the current of blood over a roughened surface, without contraction of the aperture. This is undoubtedly rare.

" Is this murmur ever produced without any mitral lesion ? One would, *a priori*, suppose the answer to this question to be in the negative. Clinical observation, however, shows that the question is to be answered in the affirmative." (? ?) He then goes on to state that he had met with two cases characterized by well-marked mitral direct murmur; in one of which no mitral lesion whatever was found after death, and in the other the lesion was insignificant. In both the aortic valves were obstructive and inadequate. He proceeds to state that when, owing to aortic reflux, the left ventricle gets filled prematurely, the auriculo-ventricular valves are approximated and

* *American Journal of the Med. Sciences*, vol. xliv., July, 1862.

oppose influx from the auricle, causing thereby direct mitral murmur. To this theory I object: such approximation occurs in the normal state of the chambers and valves, and yet no murmur occurs; no such murmur occurs in simple aortic reflux; and finally, the pressure of accumulation in the ventricle would not present sufficient resistance to the entering current to cause direct murmur.

Dr. Flint is of opinion that a pure diastolic mitral murmur may occur during the passive entrance of blood from the auricle into the ventricle, and thinks he has met such cases.

This remark is eminently just and confirmed by recent observations, as will be seen in the sequel. I have intimated by notes of interrogation my dissent from those portions of the extracts to which I take exception.

Doctor Markham devotes several pages of his work* to the discussion of the murmur of roughened or constricted mitral orifice, the quality and significance of which he correctly apprehends, but he falls into the error which Skoda and so many others had committed, of describing it as "diastolic" in rhythm, a mistake of such cardinal importance as to vitiate his entire subsequent discussion of the subject, by rendering his description useless or misleading in clinical study. Whilst admitting with Dr. Markham that systolic mitral murmur is, not unfrequently, associated with presystolic, I cannot agree with him in the opinion that such is the case in most instances, much less that it is invariably so where the constriction is considerable. Neither does my experience accord with the statement that this murmur is frequently audible over the entire precordium, or that it is associated with marked dyspnœa even in a large per-centage of cases, save at a very advanced stage of the disease, when venous congestion, anasarca, and hæmoptysis proclaim pulmonary engorgement and extravasation, and failure of the right side of the heart.

I have met with several examples of presystolic murmur which extended backwards over the entire long pause quite up to the second sound, but in no instance has it extended over the

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* *Diseases of the Heart, their Pathology, Diagnosis, and Treatment*, 1856, p. 206 *et sequent.*; and *Monthly Journal of Medical Science*, 1854.

period of the second sound, short pause, and a portion of the systole, by which in this connexion I understand the first sound, as alleged by Skoda and Markham.

Nevertheless I freely concede to Dr. Markham the merit of having been one of the first* to distinguish this from other mitral murmurs, and to recognize and insist upon its special diagnostic value, even though he failed to convey to his readers a correct notion of its rhythm, owing, no doubt, to the laxity of expression in describing the movements and rhythm of the heart permitted at the date at which he wrote.

Doctor Gairdner was undoubtedly in these countries the writer who first gave a clear and full exposition of this subject. In a remarkable paper† he not only lays down rules for the positive identification of this murmur, under the title of "auricular systolic," but contrasts it, chiefly, and very properly, on the ground of its peculiar rhythm, with all other cardiac murmurs whatever.

In his *Clinical Medicine* published the following year Dr. Gairdner reiterates the views which he had previously expressed, and gives‡ the particulars of eight cases of mitral obstruction, with three autopsies all confirmatory of the diagnosis.

Doctor Andrew, writing of presystolic murmur three years subsequently, makes the following statement :§ " So that it is safer to determine the period of the heart's action at which a murmur of this *doubtful* character takes place by its relation to the first sound, than, as is almost instinctively done, by its relation to the impulse. At the same time it must be confessed that the diagnosis of a presystolic from a systolic ventricular murmur *is one of the most difficult tasks in the physical examination of the heart, and is often all but impossible.*‖ If the murmur masks

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* In the *Edinburgh Monthly Journal* for January 1854, Dr. Markham gives a very good account of this murmur, under the name of " diastolic mitral murmur," with illustrative cases, in which the principal features of obstruction at the mitral orifice are clearly set forth ; he describes the murmur as immediately preceding the impulse of the heart, and as being indicative of mitral obstruction.

† " A Short Account of Cardiac Murmurs," *Edinburgh Med. Journal*, vol. vii., Nov. 1861.

‡ Page 599.

§ *Bartholomew's Hospital Reports*, vol. i., October, 1865, p. 33.

‖ The italics are not in the original.

and is continued through the first sound it is pretty surely systolic; if, on the other hand, the first sound is heard of natural quality, and following the murmur, it is with equal certainty presystolic and onward; but unfortunately for the auscultator the changes which occur in the auriculo-ventricular orifice or its neighbourhood, leading to the production of an onward murmur, are very frequently such as to interfere more or less with the ordinary action of the valves, and so far to alter the quality of the first sound as to render the determination of its exact commencement and duration extremely difficult."

Thus, whilst admitting in theory the distinct identity and the significance of presystolic murmur, Dr. Andrew, at the date of his memoir, regarded it as "doubtful," by reason of the difficulty, amounting almost to impossibility, of identifying it in practice.

In some lectures which I published in 1866* I insisted upon the special diagnostic value of presystolic murmur, and adduced seven cases in illustration, in three of which *post mortem* examination revealed contracted mitral orifice, confirming the diagnosis in two; in the third of these cases, which was not seen till the patient was moribund, no murmur was audible and no diagnosis was made.

Since that date I have paid special attention to this subject, and have met with fifty-six additional examples of presystolic murmur; in all these cases with one exception the diagnosis of more or less obstruction of the mitral opening was confidently made, mainly from this sign, conjoined, however, in several instances with many or all of the special symptoms which characterize the affection, to be mentioned farther on. My cases amount, therefore, to sixty-three in all. In thirteen instances out of this number *post mortem* examination of the body was made, and in all thirteen the mitral orifice was found in a greater or less degree contracted. Two of the thirteen patients who were the subjects of *post mortem* examination were moribund when they came under my notice (*vide* cases of Mrs. Quinn and Mary Armstrong) and owing to the absence of presystolic murmur the diagnosis of mitral constriction was not

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* " Clinical Lectures on Diseases of the Heart," *Medical Press and Circular*, July, 1866.

made. In the remaining eleven a positive diagnosis was made, and was verified by dissection.

Friedreich in 1867 admitted in a qualified sense the diagnostic value of presystolic murmur, declaring that the murmur associated with mitral narrowing is "sometimes" of this rhythm only. He, however, assigns to this murmur ordinarily a "diastolic" rhythm and fremitus.

In 1868 Dr. Sutton* detailed the particulars of an example of mitral narrowing which had come under his notice, and was announced by harsh presystolic murmur, which, as frequently happens, became inaudible on the occurrence of extreme debility consequent on an intercurrent seizure, in this case vomiting, and became again audible when the vomiting ceased and the patient recovered strength. It finally ceased, however, some time before death, as is the case in the majority of instances. Dr. Sutton adds that within a period of two years twelve examples of presystolic murmur had come under his notice.

Oppolzer one year later still described the murmur of mitral stenosis as "diastolic."† And in the same year Trousseau,‡ speaking of a case of cardiac disease then under observation in one of his wards, said, " Upon auscultation the cardiac lesion was revealed by a double bellows murmur, having its maximum intensity in the situation of the apex of the heart, the blowing accompanying the first sound of the heart was harsh, and that *accompanying the second* was softer. These stethoscopic phenomena were characteristic signs of valvular insufficiency, and of *constriction* of the left auriculo-ventricular orifice."§ In the foregoing extract the illustrious author of the *Clinical Medicine* distinctly intimates his opinion in regard to the "diastolic" rhythm of the murmur of mitral stenosis. In the early part of 1868 also Dr. Henry Simpson of Manchester published‖ the substance of a paper on this subject read by him before the Medical Society of that town, in which he not only fully recognized the pathognomonic significance of presystolic murmur,

* *London Hospital Reports,* vol. iv., 1867-8.

† I quote from Dr. Hilton Fagge's paper already referred to.

‡ *Clinical Medicine,* 1868, Syd. Society's edition, vol. iii., p. 396.

§ The italics are not in the original.

‖ *British Medical Journal,* May 16th, 1868.

but likewise adduced in illustration of its diagnostic value five cases, in four of which the diagnosis of mitral narrowing was made from this sign mainly, and in two was confirmed by autopsy. In the first case death occurred suddenly and before a diagnosis had been made.

Dr. A. T. H. Waters, in his work* wrote as follows on this subject: " But when the mitral orifice is much contracted it is quite conceivable that the auricular contraction may cause a murmur, which, of course, is diastolic as to time, or as some prefer to say, presystolic.

" When there is a double murmur, or even only a diastolic one, at the base, and a double murmur at the apex and towards the left axilla, although the second murmur heard at the apex and to the left may be, and probably is, of mitral origin, still such a conclusion is open to the objection that it may be the aortic diastolic murmur conveyed to the left."

The admission, contained in the last paragraph, of the possibility of confounding aortic diastolic with presystolic murmur, suffices to show that Dr. Waters had failed to apprehend the rhythm of the latter murmur, upon which exclusively its diagnosis depends.

In 1869† Dr. Hyde Salter published the details of six cases, in which a presystolic murmur had been heard, and the diagnosis of mitral narrowing made therefrom. In two of these cases the diagnosis was confirmed by examination of the body after death. In this communication he adverts to the frequency with which the presystolic murmur is terminated by a sharp and clear first sound, an observation the truth of which I can fully confirm. In some of his cases the murmur commenced immediately or shortly after the second sound, and occupied nearly the entire long pause. In reference to this peculiarity of rhythm he inquires " Does the inability of the auricle to empty itself forward provoke it to anticipate its systolic action ?" The answer to this question requires that the precise state of the auricle at the several periods of the cardiac

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* *Diseases of the Chest*, 1868, p. 363-4   In the second edition of this work (1873, p. 374) Dr. Waters fully recognizes presystolic or " mitral diastolic" murmur.
† *Lancet*, October 23rd and 30th, 1869.

cycle shall be, in the first instance, briefly reviewed. At the commencement of ventricular systole the efflux from the auricles is abruptly arrested by the closure of the auriculo-ventricular valves, but influx from the cavæ and pulmonary veins continuing, the auricles are progressively but rapidly filled and ultimately distended, a condition to which backward pressure upon the valves from the ventricles in some degree contributes. At the moment of relaxation of the ventricles the auriculo-ventricular valves are suddenly and forcibly thrown open by the removal of pressure from their ventricular surface, and the simultaneous elastic recoil of the walls of the auricles. This, I would call the passive or reactive movement of the auricles, and although due to the elastic reaction of their walls evoked by distension, it is of a forcible and energetic character, and where extreme auriculo-ventricular narrowing exists, capable of developing a murmur which must necessarily be diastolic or postdiastolic in time.

The auricles having thus partially disengorged themselves, and been thereby relieved from the reaction of distension, contract feebly and vermicularly in the direction of the ventricles, as I have repeatedly seen in experiments upon dogs, and likewise in the person of M. Groux through the thin covering of his fissured sternum. At this period likewise, which corresponds with that of the entire long pause less the presystole, a murmur may be developed in cases of extreme contraction of the auriculo-ventricular orifice, and if so developed it should be post-diastolic, or posterior to the second sound, but connected with it by continuity, and yet not strictly presystolic because anterior in time to the presystole. Murmurs of indubitable mitral origin, and corresponding in time to the latter of these periods, I have met with in a few instances (*vide* cases of Anne Coats and Thomas Doyle), and where presumably no organic lesion save that of extreme mitral stenosis existed.* I believe that herein lies the answer to Dr. Salter's query.

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* I have not met with an example of *diastolic* apex murmur in the strict sense, but Dr. Stokes has mentioned to me a case, and kindly invited me to examine it, (unfortunately I missed the opportunity of doing so), then under his care in the Meath Hospital, in which there were two murmurs, a systolic and a diastolic, at the apex, and both in Dr. Stokes' opinion purely mitral in origin.

I can, at the same time, conceive that a muscular chamber which had not been able to empty itself during the ordinary period allowed for its contraction, owing to narrowing of the orifice of exit, would anticipate the normal period of its next contraction, in proportion to the quantity of liquid remaining in it from the contraction immediately preceding, bearing in mind that the *stimulus* consists in distension. But as a matter of fact does the auricle in such case fail to empty itself during its contraction ? Certainly not, so long as hypertrophy of its walls continues to compensate for the difficulty in advance ; but ultimately, when the auricles begin to fail in this supplementary function owing to impairment of nutrition and degeneration of tissue, incomplete evacuation and consecutive premature distension must necessarily ensue. It is moreover noticeable, that where the murmur extends far backwards into the long pause the initial portion of it is faint, and that it gradually swells into a loud and harsh sound, attaining its maximum intensity immediately prior to ventricular systole (*vide* case of Thomas Doyle). This peculiarity of the murmur is more consonant with the hypothesis above stated than with that of premature active systole of the auricle.

In explanation of this phenomenon Dr. Hilton Fagge propounds the ingenious hypothesis, that owing to the tardiness with which the left ventricle is filled, and the resultant stimulus of distension applied to it, its contraction is postponed, and the whole series of events dependent upon, and consecutive to it, pushed back in a proportionate degree. Hence the second sound, in the cycle of events, is brought close up to the presystolic murmur, and the retro-elongation of the murmur is only apparent. But it may be objected that the murmur is actually and audibly prolonged in the cases contemplated, and without derangement of synchronism in the movements of the two sides of the heart. I am of opinion, as already stated, that the unusual length of the murmur in such cases is due to a *pre*fix not to a *suf*fix, and that the former is the result of abnormal effectiveness in the early or undulatory contraction of the auricle, derived from the hypertrophy of its walls.

Doctor Gee* makes some very judicious remarks on this subject, having reference, however, rather to concomitant phenomena than to the murmur itself. He very truly states that the murmur of mitral constriction is but rarely audible at the angle of the left scapula, and that accentuated pulmonary second sound fails on the establishment of tricuspid regurgitation, because then the contractile energy of the right ventricle is in some degree expended upon the reflux current. I regret to find, however, that Dr. Gee makes use of the term "diastolic" in specifying the rhythm of mitral obstructive murmur, because such laxity of expression tends to embarrass the student, and to encourage scepticism, and is scarcely excusable in an author who professes to write up this subject to the present date.

In 1870 also Dr. Paul Niemeyer† laid down as diagnostic of mitral contraction, "disordered rhythm, systolic, long, loud murmur over the apex of the heart, strong fremissement cataire, and in rare cases also short diastolic murmur." He elsewhere describes this murmur as "diastolic" in rhythm, adding that mitral stenosis is always accompanied with mitral regurgitation. From the preceding it may be concluded that in the opinion of this eminent authority the murmur of mitral stenosis is *diastolic* in time, and that the systolic murmur which he associates with it as a constant accompaniment, is that of mitral reflux, a complication of by no means constant occurrence.

In the early part of 1871 Dr. Hilton Fagge contributed a memoir " On the Murmur attendant upon Mitral Contraction."‡ To this valuable paper, which contains the fullest and best *résumé* of the subject hitherto published, I have already referred. Irrespectively of its value as a historic repertory, Dr. Fagge's memoir contains much original matter which entitles it to notice here.

The cases in which a presystolic murmur was heard and identified were twenty-six in number, and out of this number seven *post mortems* were obtained, all confirmatory of the diag-

* *Auscultation and Percussion*, September, 1870.

† *Handbuch der Theoretischen und Clinischen, Percussion und Auscultation*, band ii., 1870, quoted by Dr H. Fagge, *loco citat.*

‡ *Guy's Hospital Reports*, third series, vol. xvi., March, 1871.

nosis of mitral contraction. Forty cases are likewise given in which the mitral orifice was found contracted after death, but in which, during life, this condition was not diagnosed. This category is composed of cases in which either no physical examination of the chest, or an imperfect examination was made during the patient's lifetime; secondly, those in which a systolic bruit at the apex was noted; and thirdly, those in which no bruit whatever was detected, the patients being at the time moribund.

The first-mentioned group, or those in which the examination of the patient was inconclusive, are twenty-four in number. These are of no value whatever, and should therefore be set aside, in considering the diagnostic significance and the constancy of presystolic murmur as a sign of mitral narrowing.

The second group (excluding one in which the bruit was diagnosed as that of tricuspid reflux) includes ten cases. Some of these Dr. Fagge had himself never examined, and in at least two others he could not detect murmur of any kind. The majority are quoted from the practice of his colleagues; and considering the meagre and imperfect manner in which they are reported, I cannot attach much value to them as evidence of a want of uniformity in the acoustic sign of mitral contraction. Indeed, Dr. Fagge seems to be of the same opinion, for he does not in his summary dwell much upon them.

The cases, four in number, in which the patients were examined only *in articulo mortis*, and no murmur was heard, serve only to illustrate a phenomenon of constant occurrence, namely, the cessation or complete masking of presystolic murmur for a period varying from several hours to as many days preceding the patient's death, according to the time at which the cardiac asthenia of dissolution has set in.

Dr. Fagge justly observes: "Hitherto, I believe, no single instance has been recorded in which a presystolic murmur has existed during life, and in which the mitral orifice has not been found after death to be very decidedly narrower than usual. It has been supposed by some writers that a mass of vegetation growing from the auricular surface of the valve might obstruct the flow of blood sufficiently to give rise to such a murmur (pre-

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systolic). This may be; but I have found no recorded instance of it. Even its theoretical possibility is, I think, doubtful; for I am much inclined to believe that an essential element in the production of the presystolic murmur is the vigorous jet of blood caused by an hypertrophied auricle ; and this of course implies a chronic obstruction, such as no mere mass of vegetation would be likely to produce." He adds: "If a patient be suffering from cardiac dropsy, if the heart be beating rapidly and irregularly, and if perhaps there be in addition a loud systolic murmur, *the non-discovery of a presystolic bruit goes but a very little way to disprove, or even to render improbable, the existence of mitral contraction.*" I cannot subscribe the opinion expressed in the words which I have italicised, at least to its full extent. If a careful examination has been repeated once or oftener, the patient having been composed by rest and the warmth of bed, I do not think it likely the characteristic murmur, which I maintain is absent in veritable mitral contraction only for very brief periods and exceptionally, can be missed. It has repeatedly happened to me to *suspect* mitral narrowing at the first examination, notwithstanding the absence of presystolic murmur, and from the character of the first sound exclusively, the patient being chilled and depressed, and only recently put into bed; and on the following day to make a positive diagnosis from the existence *then* of a distinctly audible presystolic murmur. I shall have occasion further on to revert to Doctor Fagge's views in relation to other departments of this subject.

Doctor Wilks, at the same date and in the same medium, admitted in its entirety the doctrine which I have above advocated. He says: " A direct mitral bruit, however, may probably occur, not only during the contraction of the auricle, but also during the heart's diastole and pause."* In this passage it is asserted that the murmur of mitral narrowing may be in rhythm cotemporaneous with the second sound (" heart's diastole ") the peridiastole (" pause,") and the presystole (" contraction of the auricle "). And, finally, towards the close of 1871 Dr. George W. Balfour† declared his opinion that presystolic

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* *Guy's Hospital Reports*, third series, vol. xvi., March, 1871.
† *Edinburgh Medical Journal*, November, 1871.

murmur "may be conclusively accepted as a distinctive proof
of the existence of a permanent deformity, even though the
murmur itself should subsequently disappear, as it frequently
does." "The mitral orifice," he says, "is usually diaphragmatic."
In regard to rhythm, he says the murmur of mitral stenosis
often immediately follows the second sound, being sometimes
separated by an interval from the true presystolic murmur,
and sometimes including it and running right through the
periods of diastole and rest up to the apex beat. A soft dia-
stolic murmur, a pause, and a presystolic murmur in succession
are due, he thinks, to great narrowing. In the foregoing pas-
sages it is virtually admitted that the rhythm of the murmur
of mitral narrowing may be diastolic, postdiastolic, or presys-
tolic; and it is added with truth that the two former varieties,
whether single or combined, are indicative of extreme contrac-
tion of the mitral orifice. I have, however, some doubt as to
the constancy of the alleged connexion between prolonged pre-
systolic murmur and sharp first sound, on the one hand, and a
funnel-shaped mitral opening on the other.

The diagnostic value of presystolic murmur as a sign of con-
tracted mitral orifice may be judged from the record of cases
in which, mainly from the evidence afforded by it, this con-
dition of the mitral orifice has been diagnosed, and been proved
by *post mortem* examination to have existed.

Doctor Fagge gives a list of twenty-eight cases from the
following sources: Fauvel, 3; Gairdner, 4; Gull and Wilks, 1;
Rees, Gull, and Moxon, 2; Hayden, 3; Peacock, 2; Simpson, 3;
Hyde Salter, 2; Sutton, 1; Hilton Fagge, 7; a total of 28;
to which I have now to add a supplementary list of 9 cases; in
all 37.

There can be, therefore, no longer any doubt that with the
precautions against mistakes of identity previously mentioned,
this murmur is entitled to rank as pathognomonic of a par-
ticular organic lesion of the heart, namely, stenosis of the mitral
orifice; and I would add, that no other acoustic sign of itself
affords equally certain evidence of structural lesion of the
heart.

Why stethoscopists had, previously to 1843, failed to appre-

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hend this murmur, and assign to it a special significance, may, I believe, be accounted for by reference to a radical error, not even yet entirely exploded, namely, that of refusing to recognize the possibility of the occurrence of cardiac murmurs unconnected by *causal* association with either sound of the heart. Senac truly remarks: " Il en coute moins de decouvrir la vérité que de dissiper les ténebres dont on l'a enveloppée.* The universal acceptance of the traditional classification of cardiac murmurs into " systolic " and " diastolic," and the unanimous refusal of pathologists to admit any outside the domain of the sounds of the heart, led, as a necessary consequence, to a forced interpretation of murmurs occurring within the periods of silence. Hence it was that murmurs associated only by proximity with the sounds of the heart were designated as systolic and diastolic respectively.

If it had been realized that both the systole and diastole of the ventricles, and the diastole of the auricles, are continued through the periods of silence, and that the systole of the auricles occurs *only* within one of these periods, the error of assigning murmurs to the periods of sound exclusively would not have been committed. That the direct mitral murmur has been frequently, and by able and acute observers, recognized and described as diastolic in rhythm, the preceding references and quotations will have shown. This error seems difficult to comprehend, owing to the much closer relationship which the presystolic murmur holds to the first than to the second sound. In some instances, I doubt not, the mistake has been made, of assuming the first sound to be the second, owing to its remarkably sharp, clear, and clicking character,† whilst the veritable second sound was dull, and as frequently happens in these cases, partially masked at the apex, so as to resemble rather an imperfectly pronounced first sound.

In other examples, fewer in number, where the two sounds of the heart were correctly identified, I believe the error has arisen

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* *Traité de la Structure du Cœur, de son Action, et de ses Maladies*, 1749, Preface. p. xxix.

† Markham, Gairdner, and Sanders have adverted to this character of the first sound in connexion with mitral stenosis.

from the fact that the murmur occupied the greater portion of the long pause, and was therefore notably out of time with the first sound; in such cases it has been described as "diastolic," in the sense of being non-systolic. The former of these errors, namely, that as to identification of the sounds, may be avoided by applying the finger to the carotid artery whilst the sounds are being scrutinized; it will thus be found that the sound succeeding the murmur is synchronous, or approximately so, with the carotid pulse, and that it must therefore be the first, notwithstanding its unnatural character. The establishment of this fact will at once lead to a correct designation and interpretation of the murmur. The second-mentioned error cannot occur if the auscultator in the first instance recognize the possibility of murmurs within the periods of silence, and then correctly apprehend the rhythm of that under examination. The "binaural stethoscope" of Dr. Scott Alison has been recommended by Dr. Gairdner as an aid to the identification of the sounds of the heart in doubtful cases, one of the bell-ends being placed over the apex, and the other over the base of the heart. I have tried this instrument, but owing to the difficulty of keeping it steadily fixed in the ears, and the rustling noise which it consequently produced, together with the irritation to the ears thence arising, I was forced to give it up after a few trials. The "bruit de rappel" of Bouillaud, or reduplicated second sound, is, no doubt, eminently suggestive of narrowed mitral opening, because associated oftener with that than with any other condition; but it is by no means pathognomonic of this lesion. Further on I shall adduce an example (Case 86, J. Hutchinson) of this phenomenon in connexion with double aortic murmur, where mitral narrowing did not exist. (See pages 127 and 166.)

The identification of presystolic murmur depends mainly upon its rhythm, which coincides with the terminal portion of the long or diastolic pause. It commences at an interval after the second sound, usually considerable, and corresponding to at least two-thirds of the entire pause, but variable in different cases according to the degree of narrowing of the orifice; the greater that degree the more protracted will be the murmur,

and the shorter the antecedent silence by which it is separated from the preceding second sound. A distinctly appreciable interval does, however, exist in every typical case between the second sound and the murmur, whilst in no case does a period, however short, intervene between the murmur and the succeeding first sound.

The rhythm of this murmur is, therefore, accurately described as *presystolic*, a term borrowed from Gendrin, by which is implied that the murmur distinguished by it is immediately antecedent to the first sound, running quite up to, but not infringing upon that sound, and of variable length in the direction backwards, in proportion to the degree of contraction of the mitral orifice, but leaving, with rare exceptions, an interval between it and the preceding second sound.

The murmur is thus more closely related to the first than to the second sound, but in point of fact is distinct from both, and belongs exclusively to the period of normal diastolic silence.

The exceptions alluded to in the preceding paragraph render it necessary, in order to present a synoptic sketch of this murmur under its various modifications, to classify the latter under four heads, as follows:

1. The typical presystolic murmur, which answers to the preceding description, and is characterized mainly by being *prefixed* to the first sound, and separated from the preceding second sound by a long interval of silence.

2. Next in frequency of occurrence to the preceding, often succeeding to it, and representing a greater degree of contraction of the mitral orifice, is a murmur of double rhythm, or broken up into two fragments, one of which adheres as a prefix to the first sound, and represents the ordinary presystolic murmur, whilst the other succeeds the second sound, being appended to it as a *suffix*; these two fragments being separated by a brief period of silence (see Cases of M. E. Coates, M. Fergusson, and T. Doyle).

3. A protracted murmur extending backwards from the first quite to the second sound, covering the whole period of

the long pause, and representing a further development of the preceding disintegrated murmur by a bridging over of the interval of silence, and union of the two fragments, and indicating a still further degree of contraction of the orifice (Cases of T. Doyle, Jane Conroy, and Mary Brennan).

4. A murmur extending not backwards but forwards, and so completely fused into the first sound that it seems to be but the emphasized initial portion of it. This form indicates a much contracted and very rough state of the orifice (Case of Thomas Ward).

The preceding category exhibits concisely the various forms of presystolic murmur which have come under my notice. The three first are set forth in the order of their frequency, and represent, as I believe, so many stages of progressive development of one common lesion. It does not follow that every case must pass through these several stages, and exhibit in its progress the various modifications of murmur above mentioned. Death may result from various complications, or from the effects of the disease itself, in any one of these stages. The last-mentioned form is the rarest of all, and indicates, in an especial manner, a scabrous condition of the orifice, with, probably, a pendulous flap or tongue depending into it, and capable of protracting an ordinary presystolic murmur by vibration.

The transition of the first form into the second, and of the second into the third, in the progress of morbid change of the mitral orifice, may, in my view, be explained by reference to the peculiar rhythm of auricular movement, as already explained at page 206. When obstruction exists only in a moderate degree, the resultant murmur coincides with auricular systole properly so called, or with what I have designated "the momentum of auricular contraction," and therefore immediately precedes the first sound of the heart. When, in the further progress of organic change, obstruction has increased, another murmur is developed, corresponding in time to the period of passive influx of blood from the auricle, the result of the passive reaction of its walls from a state of temporary distension, on the cessation of ventricular systole. Hence, this latter murmur is

postdiastolic in rhythm, that is to say, it immediately follows the second sound. The interval of silence by which these two murmurs, or rather fragments of the same murmur, are separated, corresponds to the period of normal repose of the auricle, succeeding the passive, and preceding the active contraction of its walls. But where mitral obstruction has advanced to the last degree compatible with the maintenance of a vitalizing circulation, even this interval is bridged over by a prolongation of the postdiastolic element of murmur into the presystolic. Hence the prolonged murmur covering the entire of the long pause indicates the highest degree of mitral obstruction compatible with life.*

The *quality* of presystolic murmur is always more or less harsh, and usually decidedly so ;† it is, in its typical form, much better represented by the *bruit de rouet r r r r* of Bouillaud, than by the *bruit d'aspiration*, which he regards as pathognomonic of contracted orifice.

The harsh and disagreeable quality of the murmur is further illustrated by the frequency with which it is associated with fremitus.

I have elsewhere‡ stated that " a murmur of this character and rhythm, loudest at the left apex, not audible, or faintly so, at the base, and not transmitted, save occasionally to the left side of the lower dorsal spine, may be regarded as diagnostic of mitral obstruction."

A longer experience has afforded me no reason to alter or modify this statement in any particular.

It would be difficult, bearing in mind these specific characters,

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* The distinction above made, and the grounds upon which it is based, are, I am glad to perceive, virtually implied by Dr. G. W. Balfour in a very able article on this subject, published in the *Edinburgh Medical Journal*, for November, 1871.

† I cannot by any means agree with Hope (*opus citat.* p. 83) in the opinion that " no roughness attends the murmurs from influx from the auricles into the ventricles." See my cases of mitral obstruction, *passim*. Doctor Flint (*A Treatise on the Principles and Practice of Medicine.* third edition, 1868. p. 325) on the contrary, revokes the statement which he made in the two first editions of his book, to the effect that presystolic murmur was soft in tone, avowing the conviction forced upon him by longer experience, that the murmur in question is usually harsh and unpleasant to the ear.

‡ " Clinical Lectures," *Medical Press and Circular*, July, 1866.

to mistake presystolic murmur for any other. Nevertheless this error has been not unfrequently committed ; mainly, I believe, from failure to correctly appreciate its peculiar rhythm.

Hitherto I have met with only three sources of error in the identification of this murmur, namely : (a) Localized apex attrition murmur of presystolic rhythm, and associated with presystolic fremitus ; (b) Prolonged bellows murmur confined to the area of the apex, replacing the first sound, and extending quite up to the second sound which was associated with diastolic impulse.

In this last-mentioned group the substitutive systolic apex murmur, covering the period of the first sound and of the short pause, and touching the second sound, might readily have been mistaken for a murmur of presystolic rhythm ; and the muffled second sound associated with impulse at the left apex, quite as readily mistaken for the first (see Case of Margaret Connor). The diagnosis, however, was not difficult, and rested upon three considerations, namely, the synchronism of the murmur with systolic impulse and carotid pulsation ; secondly, the fact that at the base a second sound was heard free from murmur, which, as the stethoscope was shifted by short stages towards the apex, became gradually audible, whilst the second sound became at first faintly, and then distinctly associated with it as the apex was approached. It was manifest, therefore, that the connexion between the murmur and the second sound was entirely casual. Thirdly ; the quality of the murmur which was soft and blowing, also furnished a valuable, although an apparently trivial element of negative diagnosis.

(c) Reduplication of the first sound at the apex by resolution into its two elements, the former of which, being dull and masked, and immediately preceding the latter which was sharp and might readily be mistaken for the modified first sound of mitral contraction, is liable to lead to a mistake of identity for presystolic murmur. The differential diagnosis, however, may be readily made by reference to the *time* of carotid pulsation, which will be found to *coincide* with the *quasi* murmur ; and to the quality of the sound, which is entirely devoid of harshness, a condition, as far as I know, never associated with the murmur of mitral stenosis.

I have already stated that the murmur of mitral obstruction is harsh and unpleasant to the ear; I have never met with an example which constituted an exception to this statement, notwithstanding that Markham maintains that softness is a quality essentially characteristic of presystolic murmur.

The differential diagnosis of apex-friction sound of single and presystolic rhythm is attended with much greater difficulty, because it is usually associated with fremitus of corresponding rhythm, is loud, not transmitted, and not associated with left ventricular hypertrophy. It is, however, not steady or uniform as to rhythm, quality, or loudness; is intensified by pressure, which likewise causes some degree of pain, and is associated with a history of pericarditis, and usually with double frottement if observed early.* Dr. Chambers' test will also be found to afford assistance in a difficulty of this kind.†

Possibly, also, a murmur caused by patency of the foramen ovale, or by an aneurism of either auricle, may be mistaken for it; I have not, however, met with an example of error in diagnosis arising from either of these causes.

The principle of intersonal murmurs being thus admitted in the general recognition of the typical representative of this class, the presystolic murmur, I fail to perceive on what grounds the remaining members of the group can be logically ignored. Of *postsystolic* murmur I have met with and noted five examples. In three of these the murmur was confined to the area of the apex, and in two out of this number it was non-organic as to cause, traceable in one to excessive tobacco-smoking (case of W. Doyle,) and in the other, a female, to nervousness, and

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* In many cases of pericarditis, limited to the apex, the friction-sound is single and systolic, or pseudo-presystolic from its commencement to its extinction. A careful scrutiny of the rhythm of such, and their subsequent and early cessation, will enable the auscultator to avoid error.

† *Clinical Lectures.* If the ear be slowly and partially withdrawn from the stethoscope applied to the chest at the seat of murmur, the latter will cease to be heard if it be exocardial; whereas, if endocardial, it will be distinctly heard so long as the ear touches the stethoscope, however lightly. I have repeatedly made use of this test in doubtful cases, and have invariably found the evidence afforded by it, whether positive or negative, to coincide with that derived from more trusted sources.

associated with nervous palpitation (Case of "a woman").*. In the third case in this category (that of Catherine Hughes) there was organic alteration of the mitral valve and concomitant hemiplegia.

In one case only (No. 95, Lawrence Carolin) the postsystolic murmur was basic, and due to trivial alteration of the aortic valve, as shown by *post mortem* examination of the heart.

The fifth example alluded to above (case of Catherine Field) was one of pseudo or attrition-murmur, of single rhythm, and by its want of fixity of character, perceptible though slight variation of rhythm, and manifestly superficial location, readily distinguished from organic murmur of endocardial origin.

As to the rhythm of postsystolic murmur, it occupies that portion of the short or systolic pause immediately succeeding the first sound, the *périsystole* of Gendrin. It is always in continuity with the first sound, occasionally coinceptive with it, but extending beyond it into the brief period of silence intervening between the first and the second sound. The presystolic and the postsystolic murmur may be therefore regarded respectively as a *prefix*, and a *suffix* of the first sound. In quality likewise the two murmurs present a striking contrast ; for whilst, as already stated, the presystolic murmur is invariably harsh and vibratory, the postsystolic murmur has been, within the range of my experience, no less uniformly soft and blowing in character.

Postsystolic murmur is by no means of equal value as evidence of valvular lesion when located at the apex and at the base of the heart respectively. In the former situation it is in most instances due to atony or temporary relaxation of the walls of the left ventricle, in consequence of which, and independently of any degree of structural alteration of the valves, as I have elsewhere explained,† the parietes of the ventricle expand at one or more points under the centrifugal pressure of the contained blood at the acme of systole, and if these points happen to coincide with the attachment of the papillary

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* *Vide* " Neuroses."

† "On the Pathology and Diagnosis of Non-Organic Mitral Regurgitant Murmurs," *British Medical Journal*, December, 1867.

muscles, one or both segments of the mitral valve are of necessity partially lifted off the orifice, and a slight reflux current, commencing with the acme of systole, and therefore with, or immediately consecutive to, the impulse and the first sound, with a corresponding murmur, are the immediate result.

It is conceivable, however, that murmur of this rhythm may be produced at the mitral orifice by incipient or partial valvular disorganization, as yet not sufficiently advanced to cause systolic reflux. I have not however, met with an example which would warrant a more positive statement upon this subject.

At the aortic orifice, on the contrary, so far as I have observed, postsystolic murmur is invariably caused by valve-lesion of an obstructive character, but partial in degree, and quite inadequate to the production of valvular incompetency. In the only example which has fallen under my notice I incline to think the murmur, though postsystolic, was likewise systolic in time; in other words, that it commenced *with* the first sound but extended beyond it into the short pause; the former portion being masked by the normal sound.

I have met with only one example of *prediastolic* murmur, and in that I was afforded the advantage of a *post mortem* examination. The murmur was aortic in site, and one of the segments of the aortic valve was thick and perforated, and when closed, occupied a lower level than the other two segments (see case of Delany, disease of aortic valves.)

A pericardiac friction-sound may have this peculiar rhythm, at least seemingly, or even actually for a brief period, as illustrated by the case of P. Costello.*

In reference to the phenomena of precordial morbid sounds, pericarditis holds a position analogous to that which hysteria occupies in relation to subjective manifestations; it mimics or simulates each of them in turn, yet in such fashion that it is with no great difficulty distinguishable from them.

Postdiastolic murmur is a veritable sign of much significance and not rare occurrence. As its name implies, it immediately follows the second sound, seeming to arise out of it, and extend-

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* This murmur is not included in the scheme at page 187, because when that scheme was drawn up I had not met with the above, or any other examples of murmur of this rhythm.

ing beyond it to a variable length into the long pause. It is, therefore, the opposite of the presystolic murmur, which commences towards the close of the long pause, and extends up to the first sound.

Of murmur of this rhythm I have observed eleven examples; four were located at the apex, and seven at the base of the heart; the former were harsh, the latter soft in quality.

Out of the latter category I was afforded two *post mortem* examinations of the heart. In both these cases the murmur was aortic, and superadded to a sharp and clear second sound, and in both the aortic valves were incompetent. In one of these (Case 78, John Lalor) which yielded also a systolic basic murmur, the sigmoid valves of the aorta were rolled or shrivelled up at their free margins, so as to oppose a seam-like barrier to efflux from the ventricle, and likewise to admit, by incompetence to close the orifice, a reflux axial current during diastole.

In the second of these cases (Case 28, William H.) there was no murmur whatever save the postdiastolic, which was basic in origin; and dissection revealed a state of the aortic valves incapable of opposing an obstacle to the outflowing current, but incompetent to completely close the orifice and prevent reflux; thus, the antero-right segment was opaque and somewhat thickened throughout the "lunula," and drooped by this portion towards the ventricle, somewhat below the level of the other segments, so as to allow partial regurgitation under high pressure.

In both these cases the sigmoid valves, though incompetent to close the axial portion of the passage, were otherwise structurally healthy, and capable of yielding a sound of tension.

I am warranted, therefore, in regarding postdiastolic murmur as pre-eminently basic in location, and affording evidence, when so situate, of disorganization of the sigmoid valves engaged, by no means advanced, and permitting only partial reflux at the acme of arterial reaction, without entailing loss or even impairment of the second sound of the heart.

This murmur, at the apex, is theoretically important. In the only four cases of it so located which I have met with, there

was undoubted organic disease of the heart. In one of these (Case of Mary E. Coates) I have made the presumptive diagnosis of aggravated mitral constriction, a condition which may be readily conceived competent to give rise to a murmur of this rhythm, by causing an eddy in the current entering the ventricle by the mitral orifice at the first moment of diastole.* At this moment, as previously shown, the blood which has been accumulating within the auricle during ventricular systole, is urged forward into the ventricle by the pressure of accumulation *a tergo.* If the constriction of the orifice happen to be very considerable, a murmur may be produced by this influx, and coinciding in time with the initial portion of the long pause, the *péridiastole* of Gendrin. The murmur so produced would be not inappropriately designated an apex postdiastolic murmur.

It will be perceived, however, that I ventured on only a *presumptive* diagnosis in this case, firstly, because I had then met with only one other example, to be presently mentioned, of murmur of this rhythm at the apex; and secondly, because in neither had I the advantage of the conclusive evidence afforded by an autopsy.†

In the second example of apex postdiastolic murmur previously mentioned (Case of Eliza Donaghan) I have reason to conclude that the murmur was of a *quasi* nature, and due to pericarditis, illustrating the multiform or protean character of the acoustic signs of this disease.‡

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* Since the above was written (1870) this patient has been repeatedly examined by me, the physical signs remaining unaltered. In March, 1873, she was again admitted into hospital for dyspnœa and hæmoptysis. The murmur was then of double rhythm, *i.e.*, it consisted of a postdiastolic and a presystolic element. She is still (June 22, 1873) in hospital, expectorating large quantities of blood from time to time, and passing it alternately from the vagina, and the murmur remains as at the date of her admittance (see case).

† I have, since the above was written, met with two other examples of murmur of this rhythm at the apex, and included them in the preceding total. The "presumptive" diagnosis of that date has since become *positive,* as already stated, and the sign itself, in place of being merely suggestive, has assumed in my mind a pathognomonic significance.

‡ I do not include in this category examples of presystolic murmur prolonged backwards into the peridiastole, or in other words, of postdiastolic and presystolic murmurs conjoined. Of such the case of the boy Thomas Doyle presents a good example.

The designations *systolic* and *diastolic* as applied to murmurs, although meaning strictly coincidence in time with ventricular systole and ventricular diastole respectively, and therefore including the pauses as well as the periods of sound; have come by conventional usage to imply synchronism with the sounds only.

No doubt this limitation was due to the physiological error to which Laennec gave currency, namely, that the sounds of the heart represented the periods and the duration of its state of activity, and the pause or silence a state of complete repose of the heart. I have already endeavoured to show that there is no repose of the heart in the sense of total absence of movement, active or passive, of its chambers: whilst the ventricles are in a state of active contraction the auricles are undergoing passive expansion, and *vice versa*.

Now, although passive expansion or relaxation implies diminished disintegration of tissue, and nutritive renovation, it by no means implies absence of movement; and whilst one set of chambers is in this state of *passive* movement, the other set is in a state of *active* movement of corresponding duration. If this view be correct, and I believe it will not now be questioned, there is absolutely no period of the cardiac cycle during which two of its chambers are not in active contraction, and two others undergoing passive expansion.

The terms *systolic* and *diastolic*, although therefore critically incorrect in their usual application, as distinctive of the special rhythm of cardiac murmurs, have come, nevertheless, to acquire a definite meaning and value in pathology, and therefore may be retained with much convenience.

The precise *relationship* of murmurs to the sounds of the heart, as well as to the periods of the cardiac cycle occupied by those sounds, should receive careful attention, because of its great diagnostic value, not so much in regard to the nature and the site of the disease, as to its degree and prospective gravity.

In this sense cardiac murmurs may be conveniently classified as *substitutive* or *replacing*, and *concomitant* or *accompanying* murmurs.

The former, as superseding the normal sounds, indicate a greater degree or more advanced stage of valvular disorganization than the latter, which merely accompany them; and although the valves may be utterly disorganized at the outset owing to the extreme violence and gravity of the disease, or to the serious character of the mechanical injury which they may have sustained, and murmur alone have come to represent the normal sounds *ab initio*, the process of disorganization is usually much less rapid, and its progress may be noted with accuracy from month to month, chiefly by reference to the altered relationship of the murmur to the sounds as above indicated. I have repeatedly followed the downward progress of a case in this way from its earliest period to the end.

The pitch and quality of murmurs have been already discussed. The terms made use of by authors to designate these in their several shades remain to be enumerated, together with the causes to which they have been attributed respectively.

1. *Bruit de soufflet* (Laennec) is of high pitch, and caused, according to Laennec, by spasmodic contraction of the heart or arteries; and according to Bouillaud, by vegetations on the valves incompetent to produce obstruction. Hope maintains that this murmur, as all other organic murmurs in their degree, is due to modifications in the current, occasioning increased friction and vibration. I regard regurgitation as *par excellence* the cause of intracardial bellows murmur; and further, I can recall very few examples of obstructive murmur which were, strictly speaking, of this character, and still fewer regurgitant murmurs of the opposite type.

I am bound, therefore, to conclude, that although friction has a share in the causation of reflux murmur, it is not by any means the principal cause of that phenomenon, which, owing to the special character of softness which it all but uniformly presents, I incline to attribute to the condition which is common to all cases of regurgitation, namely, commixture of two currents moving, or tending to move, in opposite directions.

Laennec held that bruit de soufflet was the result, as above stated, of simple spasm, and did not indicate the existence of any organic lesion of the heart or arteries. Its rhythm he held

to be almost always diastolic; whereas Bouillaud maintains it is most frequently systolic. With this latter opinion my own experience coincides; it amounts to this, that the number of examples of valvular disease, including *all* stages, in which bruit de soufflet is systolic in time, vastly exceeds that in which it is diastolic. When it is borne in mind that all valvular lesions of inflammatory origin are at the outset proclaimed by murmurs of a soft and aspiratory character, it will only be necessary to add that mitral and aortic systolic murmurs conjointly, exceed in number aortic diastolic murmurs, in order to establish the truth of the foregoing proposition.

Bouillaud likewise asserts that narrowing of one of the orifices is, by giving rise to increased friction, the cause of murmur in ninety-nine out of one hundred cases where it exists. M. Piorry says not once in twenty cases. There is always associated with mitral narrowing, save where great debility of the heart exists, a murmur, but of a harsh character.

Under the generic title of bruit de soufflet, Laennec included four species, namely :—

(*a*) *B. de soufflet, properly so called.* This is associated by Bouillaud with stenosis and indurated valves arising from fibrous or cartilaginous, rather than from calcareous, change.

(*b*) *B. de scie,* which is attributed to the same cause, with calcareous induration, by Bouillaud.

(*c*) *B. de râpe.*

(*d*) *B. de lime à bois.* The two last are caused by great constriction, with calcareous rough and uneven valves, and strong contraction.

To the preceding Bouillaud adds, as a fifth species of the bellows murmur,

(*e*) *B. de roucoulement.*

I cannot perceive the slightest resemblance between the *genus* and the alleged *species* above mentioned, with the exception of the first. On the contrary, I regard them as in no respect german, and even as representing opposite pathological conditions; the former, softness of texture, smoothness of surface, and inadequacy of valves; the latter, rigidity or calcification of tissue, roughness of surface, and in most instances obstruction of valves.

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2. *B. sec dur parcheminé*, associated with thickening and rigidity of the valves, especially the mitral (Bouillaud).

3. *B. d'aspiration*, indicative of mitral constriction, according to Bouillaud. I have already stated my opinion that this does not represent the murmur of mitral narrowing.

4. *B. de scie, s s s s* prolonged, (do.).

5. *B de rouet, r r r r* prolonged, (do.).

This last I believe fairly represents the usual quality of presystolic murmur, as already stated.

6. *B. de râpe* (do.).

7. *B. de lime* (do.).

8. *B. de sifflement musical* (do.).

9. *B. de fusée* (do.).

Murmurs of the quality of any of the six last mentioned arise from stenosis with indurated valves, according to Bouillaud.

10. *B. de flot* (do.).

11. *B. de miaulement* (do.), in mitral narrowing.

12. *B. de tintement métallique* of Bouillaud, and attributed by him to ossification of the heart or pericardium. I have met with three examples which correspond with the description of this murmur given by Bouillaud, and which, I think, would be more appropriately named *B. de tintement gastrique*, because in my opinion due to gastric resonance imparted to the right ventricle of the heart through the diaphragm. It is not always systolic, as stated by Bouillaud; of three examples of it which have come under my notice, it was postdiastolic in two, and systolic in one only. In two of these it was strictly confined to the sixth and seventh costal cartilages about one inch to the left of the sternum, and in the third to the base of the xyphoid cartilage. The resonance was peculiar and unmistakeably gastric, and consisted rather in an after-tone or echo than an accompanying ring.

13. *B. de timbre enroué, âpre ou fêlé.* (do.), depends upon a tumid, soft, and spongy state of the valves.

14. *B. rauque* (do.). These two last-mentioned murmurs represent endocardial murmurs of the lowest pitch.

15. *B. de frôlement* (do.), indicative of earliest stage of peri-

carditis, in which the serous surfaces are dry, and as yet free from false membrane; but when it is very loud and decided, resembling the bruit de râpe or bruit de scie, there is already false membrane. This murmur resembles the noise made by the rumpling of silk or new paper; it is usually double, more pronounced during systole than during diastole, superficial, as if produced close to the ear, and diffused. In all these particulars it differs from valve-murmur.

16. *B. de cuir neuf* (Collin), also diagnostic of pericarditis, but in the stage of false membrane which has become dense, firm, elastic, partially-adherent, and is subjected to strong tension; it is less common than the last-mentioned, and especially accompanies ventricular systole.

17. *B. de raclement* (Bouillaud), due to grating of rough and hard surfaces, and isochronous with the action of the heart, or of double rhythm; it indicates osteoid plates in the pericardium.

The three last-mentioned *quasi*-murmurs are modifications of the attrition sound of pericarditis, for which the title of "murmur" is highly inappropriate; whenever a veritable bellows murmur is associated with pericarditis Bouillaud regards it, and very correctly in my judgment, as evidence of the complication of endocarditis.

18. *B. de frottement sec* (Gendrin), of systolic and diastolic rhythm, superficial, confined to the precordium, and due to the forcible rubbing of the opposed surfaces of a healthy pericardium upon one another during palpitation.

19. *B. de va-et-vient* (Gendrin), due to inadequacy of the aortic or pulmonic valves, and being in rhythm, according to Gendrin, *périsystolic* and *prédiastolic*. This is the "to and fro" murmur of modern writers, of which the former element is undoubtedly systolic, and the latter diastolic in rhythm.

I have excluded from my classification murmurs of alleged prediastolic rhythm; but, as already stated (p. 220), I have recently met with a veritable example of murmur of this rhythm, and had the advantage of associating it with a definite valvular lesion by *post mortem* examination of the body.

I shall now advert to other and more special characteristics

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commonly made use of to distinguish murmurs. But inasmuch as these features imply a foregone conclusion as to the special site and effect of the structural change which has given rise to the murmur, they have not been considered in connexion with those already discussed, which *lead to* rather than *assume* a specific diagnosis.

Of such a character are the designations *mitral, aortic, tricuspid,* and *pulmonic;* which become still more specific and precise in conjunction with the terms *direct, obstructive,* or *afflux ;* and *indirect, regurgitant,* or *reflux.*

Thus, a substitutive regurgitant mitral murmur would mean the same thing as a substitutive systolic left apex-murmur.

The latter, however, would be the preferable formula to adopt; because, as already stated, it leads to, rather than anticipates a conclusion.

As previously mentioned, Laennec held that bruit de soufflet was proximately caused by simple spasm.

Gendrin regarded friction as the sole cause, and accordingly named the most simple murmur *bruit de frottement.*

Reflux is, *par excellence,* the cause of bruit de soufflet, as M. Filhos long since urged.[*]

Bouillaud considered narrowing of one of the orifices, by increasing the friction between the orifice and the blood, as the principal cause.

According to the same authority, the second cause of B. de soufflet, in the order of numerical frequency, is fibrinous concretions engaging in the orifices.

Third. Hypertrophy and dilatation of the left ventricle consecutive to aortic stenosis.

Fourth. Tumefaction of valves, as in an early stage of endocarditis; vegetations and cartilaginous or calcareous plates upon them without alteration in size of orifice.

Fifth. Adhesion of valves to adjacent walls of ventricle, and consequent inadequacy.

Sixth. Dilatation of any of the orifices, and valvular incompetency arising therefrom.

Seventh. Hypertrophy with dilatation of the left ventricle.

* *Dissertation Inaugurale.*

Bellows murmur likewise occurs occasionally after fatigue, effort, or moral emotion, in connexion with violent action or palpitation.

Eighth. Chlorosis and anæmia; murmur of a faint character.

Ninth. A temporary bruit de soufflet after great loss of blood.

Bouillaud says that reflux murmur, aortic or mitral, is single, whilst that of constricted orifice is frequently double.

The former proposition is all but universally true; as valvular disease, whether mitral or aortic, *primarily* regurgitant, very rarely assumes in the progress of organic change the character of the twofold lesion. And seeing that, in tissues which are themselves the seat of chronic disease, wasting is the all but universal law, this truth might have been theoretically accepted.

In regard to the second assertion, viz., that the murmur of constricted orifice is frequently double, I find that such was the case in nearly one-third (eighteen out of sixty-three) of my cases of mitral narrowing; whilst, of thirty-three cases of aortic obstruction, the murmur was double in twenty-six.

I have not met with an example of adhesion of the valves to the walls of the ventricle, (No. 5) as a cause of valvular incompetency. Of valvular inadequacy arising from dilatation of the orifice (No. 6) without structural disease of the valves, I have observed and recorded several examples, in which the lesion was either aortic or tricuspid, but very few indeed in which it had its seat at the mitral orifice.

The examples of bellows murmur (No. 7) attributed to fatigue, moral emotion, palpitation, etc., I regard as instances of a dynamic murmur due to atony and temporary yielding of the walls of the left ventricle, with consequent reflux.*

Laennec says :† " Bruit de soufflet may accompany the diastole of the heart, and that of the arteries, and is so associated with them that it replaces and supersedes entirely their natural sound, so that *at each diastole the ventricle, the auricle, or the artery* in which the phenomenon takes place, yields distinctly a bellows sound which ceases during systole."

In the words which I have italicized, the illustrious Laennec

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* See Paper by the author, *British Medical Journal,* December, 1867.

† *Traité de l'Auscultation Médiate,* tom. ii., p. 422.

clearly implies his acquaintance no less with murmurs of reflux than with those of afflux; and no less plainly intimates the distinction between systolic and diastolic murmurs, although not admitting them by name, and attaching undue importance to the less effective movement of diastole. Thus, for example, the auricular diastolic murmur of Laennec, coinciding with ventricular systole, would be a systolic murmur in modern terminology, and therefore either mitral regurgitant, or aortic obstructive. The same may be said of his arterial diastolic murmur; whilst his ventricular diastolic murmur would be, according to modern interpretation, either aortic regurgitant, or mitral obstructive; the latter being incorrectly regarded as diastolic.

Notwithstanding the indubitable priority of Laennec in promulgating, at least impliedly, the doctrine of regurgitant murmurs as above shown, Hope claims to have been, in his work published in 1831, the first to describe and invite special attention to murmurs of regurgitation.

No doubt he described them more specifically, and with greater precision than did Laennec; but to assert, as he does, that the French authors prior to himself, including Laennec, were unacquainted with the phenomenon of reflux as a cause of murmur, seems somewhat in excess of what facts warrant.

The *area of apex murmurs* may be represented by a circle of about three inches diameter; or rather by a series of concentric circles or zones, of which the external would possess this diameter, and the central, corresponding to the area of apex pulsation, a diameter of about one inch. The centre of this circle, at which the pulsation of the apex may be detected by the hand placed flat upon the precordium, is located in the fifth intercostal space of the left side, one inch inside the nipple line, two inches from the edge of the sternum, and about the same distance below the nipple-level. The localization of this area depends entirely upon that of the apex itself, which is subject to variation dependent upon (*a*) the position of the body, (*b*) the relative volume of the two ventricles, and (*c*) the volume and condition of adjacent organs. The position of the apex may be ordinarily determined by the application of the hand as already mentioned; this may be accomplished most readily and

satisfactorily by means of that portion of the palm correspond-
ing to the roots of the fingers, which possesses a tactile sensi-
bility of great delicacy in regard to impressions of this kind,
due, probably, to the great extent of surface presented, by which
the advantage of comparison with the adjacent cutaneous sur-
face is obtained. In the event of no apex pulsation being
discoverable, as occasionally happens, for example in great
obesity and in the subjects of weak-acting or fatty heart, a
slight inclination of the body to the left side, or placing it in
the prone position, may enable the examiner to detect the apex-
beat. Where even these measures fail, the point of the thoracic
surface corresponding to the apex may be easily determined by
fixing that of greatest intensity of the first sound, which coin-
cides with the apex-point, and may be quickly ascertained by a
practised auscultator. It must be remembered that the position
of the apex varies slightly with that of the body, shifting about
half an inch to the right or left side with a corresponding
inclination of the body, when not opposed by strong and
extensive pericardial adhesion, or by increased volume of the
subjacent lung. In cases of hypertrophy of the left ventricle
with or without dilatation, but especially in the former, the apex
is displaced to the left, and may be found pulsating at any point
within a range extending two inches outside the nipple line.
Or the displacement of the apex may be in the direction down-
wards and outwards beneath the sixth rib; or even into the sixth
intercostal space and outwards to a distance of half an inch to an
inch external to the nipple line. The former of these displace-
ments, which are usually due to mitral regurgitation, are the
consequence of globular enlargement of the left ventricle; whilst
the latter, which, when not caused by displacement of the heart
in its totality, are pathognomonic of aortic reflux, indicate
elongation as well as increased diameter of the left ventricle.
Hypertrophy with dilatation of the right ventricle may alone
suffice to cause partial displacement of the apex to the left; but
inasmuch as, unassociated with hypertrophy of the left ventricles
it is usually dependent upon pulmonary emphysema, the apex-
beat is rarely perceptible, being masked by this affection. Being
generally preceded and accompanied by left ventricular hyper-

trophy, increase of volume in the right ventricle contributes in a proportionate degree to the left lateral displacement of the apex, which is justly attributed in greatest part to the affection of the left side of the heart.

General pulmonary emphysema has the effect of displacing the heart downwards and inwards, and therefore of abolishing its pulsation in the ordinary site. In such cases the heart may be felt pulsating in the scrobiculus cordis, and its sounds heard most distinctly at the ensiform cartilage. This anomaly is explained by the state of the lungs and the depression of the diaphragm.

The apex of the heart may be displaced to the right or to the left in various degrees by liquid effusion into the opposite pleura; and by cirrhosis or other contraction in the volume of the lung it may be drawn towards the affected side. I have seen the heart displaced upwards and inwards, and pulsating beneath the left clavicle, in a case of inferior thoracic aneurism (see Aneurism, Case of Fay); and I have likewise found it displaced upwards and outwards by a circumscribed purulent accumulation in front of the liver, connected with and derived from an empyema of the right side.

A murmur audible only within the area of the apex, and with greatest intensity at the point of apex-pulsation, would be of mitral origin, whether presystolic, systolic, or postsystolic. Presystolic murmur is, in the majority of cases, strictly confined to the apex-area. Systolic murmur, when of moderate strength, is likewise limited to this space ; but a loud murmur of systolic rhythm, and irrespectively of its quality, is transmitted upwards in the left axillary line to an extent proportionate to its intensity, and is likewise audible in the left back near the inferior angle of the scapula. Postsystolic apex-murmur is usually soft and faint, and its diffusion is limited to the area of the apex. The two former are usually, but by no means invariably, associated with accentuated second sound in the pulmonary artery, as first pointed out by Skoda.* This phenomenon, itself directly dependent upon hypertrophy of the

* *A Treatise on Auscultation and Percussion*, translated by Markham, 1853, p. 232-3.

right ventricle, will of course be absent in the early stages of both lesions previously to the increase in volume of the right ventricle; and throughout the disease in those cases in which the narrowing of the orifice or the incompetency of the valves exists only in a minor degree, although sufficient to give rise to murmur.

The cause of mitral murmur is usually structural alteration of the valves or of the orifice, competent to oppose in greater or less degree the flow of blood from the auricle into the ventricle, or to prevent the closure of the valves in a degree sufficient to preclude reflux.

This structural alteration is, in the great majority of cases, if not in all, of inflammatory origin, and the direct result of antecedent endocarditis, which manifests a predilection for invasion of the fibrous structures in and about the orifices; it consists in interstitial cell-proliferation and deposit upon the valves and tendinous zone. By this means the valves are thickened, uniformly or at intervals, and not unfrequently attached to one another, or rolled up into a dense mass, offering at once obstruction and rendering closure impossible. In such extreme cases the attached chordæ tendineæ are likewise thickened, and elongated or shortened according to the fixed position of the valves.

When the valves are thus utterly disorganized, not only are their functions annulled in regard to the flux and reflux of the blood-stream, but likewise with respect to the production of tension-sound; hence, in such cases, the systolic or reflux murmur is substitutive; in other words it replaces the first sound, or by depriving it of the valvular element, reduces it to the condition of a simple impulse-sound. In less extreme cases the lesion may consist in a nodular or wart-like deposition or outgrowth on the surface or edge of the valve-segments or tendinous ring. If on the auricular surface and at the attached margin of the valve, such formation may simply interrupt the passage of the blood in its normal course, and give rise to direct, obstructive, or presystolic murmur only; the unaltered lunulæ of the valves being competent to effect occlusion of the orifice in systole, and yield a tension-sound. Hence, in such cases, which constitute only a very small per-centage in obstructive

lesion of the mitral orifice, the first sound is normal and forms a natural pendant to the murmur. In most of these cases, however, the valves are thickened and incapable of yielding sound, which must be due to tension of the tendinous chords. If, on the other hand, the lunulæ have undergone such structural alteration, whether by thickening, excrescence, or shrinking, as to be no longer capable of effectually closing the orifice, but without opposing in any degree the efflux of blood from the auricle, a systolic murmur only, accompanying or substitutive according to the structural condition of the valves, will be the result. An eddy may be produced in the blood-current from the auricle into the ventricle by a minute outgrowth from the auricular surface of the valve, or by a flake of fibrine whipped from the blood and entangled in the meshes of its free margin, and an afflux-murmur of presystolic rhythm be so developed. In such a case, whilst the murmur would afford indubitable evidence of the position of the mechanical impediment, the symptoms, whether subjective or objective, would be null, owing to the absence of any sensible impediment to the circulation; and if the valve-lesion be not progressive, as not infrequently is the case, it may exercise no influence upon the duration of life, and the apparent anomaly would be presented, of a person with organic disease (?) of the heart, as declared by physical evidence, utterly unaware of its existence, suffering no inconvenience of any kind from its presence, and living to the full period even with enjoyment under it. Examples of this kind are comparatively numerous, and afford a striking illustration of the impropriety of announcing openly " disease of the heart,"—a phrase pregnant with the most alarming significance to the uninitiated—in all cases indifferently from physical evidence alone.* Calcareous degeneration of the valves and adjacent structures whether normal or adventitious, although of more

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* I feel bound, however, to state, that although I have met with several examples of presystolic murmur (vide cases passim) of an imperfectly pronounced character, but with the peculiar rhythm, in persons enjoying fairly good health, and even the pleasures and gaieties of society, and have repeatedly examined them, I have no post mortem evidence to produce in confirmation of the above doctrine. I adhere to the opinion, therefore, that the characteristic harsh presystolic murmur is pathognomonic of mitral narrowing.

frequent occurrence at the aortic orifice, is occasionally met with at the mitral opening. The subjects of it are usually persons of middle or advanced age and gouty diathesis; the youngest persons in whom I have had demonstrative evidence of its existence were aged twenty-three and twenty-six years respectively (cases of P. MacD. and A. M.). In this form of valvular lesion the endocardial investment of the cretaceous masses has been destroyed; and the subjacent surface,·rough by the mode of aggregation of the adventitious particles, is presented directly to the blood, and gives rise to a harsh, filing, or rasping murmur.

As to the pathology of this form of valvular degeneration, I incline to the opinion that it is not necessarily, or even usually, preceded by acute inflammation of the endocardium. In several examples within my personal experience it has been found where there was no history of rheumatism, gout, or acute affection of the heart. In these cases I am forced to regard the valvular lesion either as a primary degeneration of a special structure, and belonging to the same category as atheromatous transformation of the arterial coats; or as the result of degenerative change in the products of chronic inflammation.

The inflammatory deposits in or upon the surface of the valves not unfrequently, in chronic cases, assume a cartilaginous density and elasticity, and a pseudo-cartilaginous structure. The endocardial covering is, however, unbroken, and the surface of the thickened and irregular valve therefore smooth. Hence, the resultant murmur is the reverse of harsh in quality.

An occasional though rare cause of mitral systolic or regurgitant murmur is rupture or disintegration of one or more of the chordæ tendineæ, in consequence of which the corresponding valve-segment is reversed in some degree at the acme of ventricular systole, and partial reflux takes place. The reversion of the valve, unless in the event of detachment of several tendons, an occurrence of extreme rarity, must be incomplete, as each segment receives a group of tendons from both papillary muscles. Irrespectively of the history, I am not acquainted with any specific character either in the symptoms or physical

signs by which lesion of this form may be differentially diagnosed. (See Case 24.)

The anatomical position of the root of the aorta would seem to imply, that the point of maximum intensity of aortic murmurs should coincide with the left third sterno-costal articulation and the adjacent portion of the margin of the sternum; but as the vessel is here overlain by the infundibulum of the right ventricle, the sounds emitted from it, whether normal or morbid, are more distinctly heard somewhat to the right, or at mid-sternum. This point is, however, liable to variation; thus, in pulmonary emphysema engaging the anterior edges of the lungs, the heart is displaced downwards with the diaphragm, and the point of maximum aortic distinctness is correspondingly depressed. Similar displacement results from great hypertrophy of the heart, simply by its weight. Enlargement of the left lobe of the liver may, on the contrary, displace the heart upwards, and elevate the aortic point to a corresponding extent. The transmission of aortic as of other murmurs is governed by the law of current-conduction; hence those of systolic rhythm may be heard, with scarcely diminished intensity, from the mid-point of the sternum upwards along the middle line and right margin of that bone to the level of the first intercostal space, and thence upwards in the course of the great cervical vessels, beneath the right sterno-clavicular articulation, into the right side of the neck; and usually also, but with less distinctness, across the lower portion of the manubrium sterni to its left margin, and upwards into the neck and beneath the clavicle in the course of the left carotid and subclavian arteries. Systolic aortic murmur of extreme loudness and special harshness, if associated with general atheromatous transformation of the arteries, may be heard even in the primary and secondary arterial trunks throughout the body. I have heard such a murmur not only in the descending thoracic and abdominal aorta, but likewise in the brachial, radial, iliac, and femoral arteries. This murmur is also transmitted downwards as far as the ensiform cartilage, but is very faintly audible at that point.

Diastolic aortic murmurs have a much more limited range of diffusion; they are distinctly audible at the xyphoid cartilage,

and over the sternum to the right of the mesian line as high as the second costal cartilage; but rarely, according to my experience, in the aortic arch beyond the first curvature, and never in the carotid or subclavian arteries except when associated with aneurismal dilatation of these vessels.

Doctor Gairdner asserts[*] that diastolic aortic murmurs are invariably propagated into the arteries of the neck, "though sometimes very faintly." I have not met with a single example of diastolic aortic murmur which was audible *even* faintly in the carotid or subclavian arteries; whereas, systolic aortic murmurs may invariably be heard in these vessels.

But aortic murmurs, both systolic and diastolic, are occasionally audible at the apex with much distinctness, and in other directions likewise far beyond the limits of the aortic area. I have heard them beneath both clavicles, and even in the left back. In such examples, which are strictly exceptional, the left ventricle is not only hypertrophied but dense and firm in texture, and acts with unwonted vigour; and both it and the osseous framework of the chest serve as conducting media of sound. I cannot conceive a careful observer committing an error of diagnosis in such cases, due attention being given to the point of maximum intensity of the murmurs, and to the increasing faintness with which they are heard as the stethoscope is gradually shifted from this point, as a common centre, to the utmost limits of diffusion. Again, the identity of the systolic aortic murmur may be always crucially determined by its transmission into the carotids, a feature which distinguishes it from all other valvular murmurs. A diastolic murmur at the apex is presumably *not* mitral. I cannot recall from my personal experience a single example of mitral murmur strictly diastolic in time.[†]

A pericardial friction sound of diastolic rhythm, and closely simulating an endocardial murmur, may, no doubt, be heard at the apex, and even be confined to that situation (case Eliza Doyle); but the identification of this by means of its special characteristics, and above all, its rhythmical variations from day to day,

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* *Edinburgh Medical Journal,* vol. vii., 1861-2, p. 451.
† See foot note, page 206.

cannot be attended with much difficulty. A postdiastolic aortic murmur is not unfrequently heard at the base, to which it is usually limited; it is invariably soft and blowing, and, as its designation implies, it succeeds without interval, or by continuity, a second sound scarcely if at all differing from the normal sound in character and intensity.

I have already expressed my opinion as to the special pathological significance of this murmur; to the effect that it implies a partial, and usually an axial reflux at the orifice of the aorta, the valves being either in a very slight degree altered from the normal state, or structurally sound. I regard this murmur, then, as evidence either of a transitional state of the valves, an early stage in the process of valvular disorganization, or of dilatation of the aorta without valvular disease. It is therefore of much value as an aid to precise diagnosis, and in determining the probable duration of life.

Tricuspid murmurs, or those having origin at the right auriculo-ventricular opening, though much less common than murmurs at the mitral orifice, are by no means infrequent. They are usually systolic in rhythm, soft in character, and most distinctly audible to the left of the middle line of the sternum at the level of the sixth costal cartilage, but diffused throughout the tricuspid area, beyond which they are never heard. A presystolic murmur is occasionally developed at the tricuspid orifice; its rhythm is identical with that of mitral origin; it is, however, less loud, as might be inferred from the relative thickness and strength of the two auricles, and its point of greatest intensity corresponds to the fifth intercostal space one inch to the left of the sternum. It indicates narrowing of the tricuspid orifice.

Of this lesion I have met with only three examples (cases of P. McD., Mary M., and Michael F.). In all three, tricuspid was associated with mitral constriction; and in one of these the latter lesion alone was diagnosed, the supplementary diagnosis of tricuspid narrowing was not made, although it was remarked and specially noted that presystolic murmur was distinctly audible near the left margin of the sternum. In the second and third cases the twofold lesion was diagnosed.

Enlightened by my experience of the two former cases, in which the body was examined after death, I would in future regard the existence of two centres of presystolic murmur with or without fremitus, viz., at the apex, and somewhat to the right of that situation, in conjunction with marked systemic venous engorgement, as evidence of the double lesion of mitral and tricuspid stenosis. From these premises the diagnosis in the third case was made.* I have never heard a diastolic murmur which I could confidently refer to the tricuspid orifice.

The *area* of tricuspid murmurs is of an irregularly triangular figure, having its base at the level of the ensiform cartilage, and extending from the middle line of the sternum to a point in the left fifth intercostal space about an inch to the inside of the apex. The vertex of this triangle would correspond to the lower edge of the left fourth costal cartilage near the sternum ; and its sides to two oblique lines connecting this with the extremities of the base. Thus, the lower third of the left margin of the sternum, and the inner portion, to the extent of about an inch and a-half, of the fifth and sixth costal cartilages, and fifth intercostal space of the left side, together with a somewhat less extent of the fourth intercostal space, would be included in this area.† It therefore overlies the inner half of the mitral area ; but, whereas murmurs of the latter origin have their point of maximum intensity *at* the apex, tricuspid murmurs are loudest, and perceived to be closest to the ear, from one inch to two inches internally to that point.

The foregoing description and mapping of the area of tricuspid murmurs are based upon a careful study of the three cases already mentioned. I believe they will be found in practice to define pretty accurately the limits within which tricuspid murmurs are confined, although differing in many particulars from the descriptions of previous writers. The space thus included would be a nearly equilateral triangle, and the point of greatest

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* In cases of the twofold lesion, the loudness, and usually also the quality of the murmur, will contrast strikingly at two points equidistant from, and to the right and left of, that of apex pulsation.

† See Plate Fig. II.

intensity of tricuspid murmurs would be, *quam proxime*, the centre of this space.

Of murmur arising at the orifice of the pulmonary artery I am acquainted practically with only one form, namely, that of systolic rhythm; of this I have met with eight examples, six in females, and three in males. In the former it was associated with anæmia and leucorrhæa without a single exception, and in the greater number with menorrhagia likewise (cases of Anne McK., R. E., Anne C., Anne S., Mary McC., and Miss C.). Most of these females were under twenty years of age, and one only was over forty. Two of the male patients were nervous and under twenty years; one was addicted to masturbation; and the third was the subject of carcinoma of the mediastinum engaging the root of the pulmonary artery.

As to the quality of the murmur, it was harsh in two instances, and in the remaining cases it was soft.

The point of maximum intensity, the area of diffusion, and the transmission of this murmur are of great value diagnostically.

The area of pulmonic murmur corresponds to a circle of about two inches in diameter, covering the inner portion of the second intercostal space of the left side, and adjacent portions of the second and third costal cartilages, and also, to a slight extent, the edge of the sternum.

Beyond the limits of this circle murmurs of pulmonic origin are rarely audible. Those which have occurred in my experience were in every instance limited to it; they were not traceable upwards towards the clavicle and into the neck as are those of aortic origin, and were not audible in the left back as are mitral systolic murmurs of much force, The rhythm of this murmur might readily lead the observer to mistake it for systolic murmur in the aorta; but its *abrupt* termination in the direction upwards at the level of the middle of the second costal cartilage, its not being audible to the right of the mesial line of the sternum, and its non-transmission into the carotids, sufficiently distinguish it from the latter, irrespectively of the difference as regards the point of maximum intensity in the two cases. From mitral murmurs the last-mentioned difference will serve as the chief distinction, but in a less degree

the circumstance that mitral murmurs when loud may be heard likewise in the left back will serve to distinguish it.

In none of the cases which have come under my notice, with one exception, have symptoms of organic lesion existed; the patients, with this exception, were all anæmic, venous murmur existing in the greater number, and in several the cardiac murmur ceased under ferruginous treatment. Notwithstanding, therefore, that in only one of these cases (Case 66) have I had an opportunity of examining the heart by dissection, I feel warranted in concluding that the murmur in all the others was hæmic.

Hope has never met with an example of valvular lesion in the pulmonary artery,* nor does he allude to hæmic murmur in this vessel.

Contraction of the orifice of the pulmonary artery is a usual concomitant of permanent patency of the foramen ovale; but, in such cases, the diameter of the vessel being, by a gradual process of accommodation, simply adapted to the diminished volume of blood passing through it, and no disproportion between the vessel and the blood-stream thereof existing, murmur is not produced. It is conceivable, however, that systolic murmur in the pulmonary artery may result from compression of that vessel by a tumor of any kind. Of this case, 66 was an example.

Hope has reported one case of diastolic murmur in the pulmonary artery due to dilatation of the vessel and consequent inadequacy of the valve. He states that the proportion of valvular disease on the right and left sides of the heart is as 1 to 16 or 20, and that this lesion is by much less frequent in the pulmonary artery than at the tricuspid orifice. Walshe places cardiac murmurs of organic origin in the following order of frequency, viz., mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.†

Other causes of intracardial organic murmurs may exist irrespectively of valve-lesion. Thus, murmur may be due to

* *A Treatise on the Diseases of the Heart*, third edition, 1839, p. 385.
† *Diseases of the Heart*, third edition, p. 108.

patency of the foramen ovale, to aneurism of one of the chambers of the heart, or to aneurism of the aorta or pulmonary artery communicating with one of these chambers. I have met with three examples of unclosed foramen ovale, in each of which I have had *post mortem* evidence of this organic defect. Two were past the middle period of life, and one was a child. In one of the former (Case 72) there was apparent hyperoxidation of the blood, the cheeks and lips being florid, and no tendency to coma. Fatty degeneration of the heart and considerable hypertrophy of the left ventricle were found to exist, and likewise patency of the foramen ovale, but no valvular disease. In this case no cardiac murmur of any kind had existed.

In the remaining two cases there was cyanosis, and in one of these (Case 71) systolic apex-murmur. In this latter case no valve-lesion existed, but the muscular tissue of the heart was in an advanced state of fatty degeneration; and to the weakness and yielding of the walls of the left ventricle during systole, arising from this cause, I am disposed to attribute the murmur of mitral reflux.

In the third case (No. 73), no murmur whatever existed. I cannot, therefore, speak from personal experience of murmur at the foramen ovale. Nevertheless, I can admit it theoretically, and, as I have elsewhere* explained, its rhythm in my view would be presystolic.

Of aneurism of the several chambers of the heart, and of the aorta communicating with one or other of these, Dr. Thurnam has given numerous illustrative examples.† In regard to rhythm there is a dearth of reliable evidence; but, guided by my personal experience of two fully observed cases (Nos. 69 and 70), I believe it may be stated, generally, that in the former series the murmur coincides with the diastole of the chamber whence the aneurism springs, and in the latter that it is both systolic and diastolic.

I cannot agree with Dr. Walshe‡ in the opinion "that excess of force of propulsion of naturally constituted blood would

* "Lectures on Diseases of the Heart," *Medical Press and Circular*, July, 1866.
† "Memoirs on Aneurism of the Heart," 1838; and "Spontaneous Varicose Aneurism of the Aorta," *Med. Chir. Transact.*, 1840, vol. xxiii.
‡ *Diseases of the Heart*, third edition, 1862, p. 283.

seem capable of generating direct murmur;" nor have I met with a case of hypertrophy in which mitral regurgitant murmur occurred during palpitation by disturbance of the action of the papillary muscles, as Dr. Walshe surmises. Such murmurs, he adds, are fugacious. I have met with murmur answering to this description, only in cases of weakened heart; as in those addicted to excessive tobacco smoking, or to masturbation.

Finally, I cannot recall a single authenticated example, in my own experience, of murmur at the aortic orifice arising from mere derangement of proportion between that orifice and the left ventricle, by dilatation with hypertrophy of the latter, as alleged by Walshe.* I have invariably found at the orifice, in such cases, some structural alteration sufficient to account for the murmur.

Doctor Purser thinks that where the left ventricle is greatly dilated, as in permanent patency of the aortic valve, a murmur of mitral regurgitation may arise from valvular incompetency produced by *relative* shortening of the chordæ tendineæ; that is, by the absence of lengthening of these chords proportionate to the expansion of the cavity of the ventricle† Such an occurrence is conceivable, but the condition mentioned would be readily detected on *post mortem* examination of the heart, more especially as the absence of lesion of the valves would necessarily lead to a careful scrutiny as to the cause of the murmur. I have occasionally met with examples of mitral inadequacy, as indicated by murmur, where neither valvular lesion nor dilatation of the orifice existed. This I have endeavoured to explain in another way (*vide antea*, p. 219), but I have never witnessed the pathological combination mentioned by Dr. Purser.

Doctor W. T. Gairdner has made some original and valuable observations on organic murmurs and their combinations.‡ He admits postsystolic murmur ("ventricular systolic") but does not make a distinction between it and murmur of systolic rhythm. He likewise virtually admits postdiastolic murmur, which, if valvular, is always due to blood entering one of the

* *Opus citat*, p. 284.
† *Irish Hospital Gazette*, March 15th, 1873.
‡ *Edinburgh Medical Journal*, vol vii., November, 1861.

ventricles " either from the auricle or the artery ;" in the latter case indicating reflux.   In this, as in the former instance, however, he fails to distinguish between murmurs synchronous with, and those succeeding the sound.   He denies entirely prediastolic murmurs.

I have met with only three examples of left auriculo-ventricular postdiastolic murmur (cases M. Coates, T. Doyle, and M. Ferguson); and this, as already stated, I believe to be due to extreme mitral narrowing.

As to the combination of mitral murmurs, I hold that of presystolic and postsystolic (" auricular-systolic and ventricular-systolic"), or that of one " running up to, with one succeeding or running off from the first sound," which implies that the first sound is perfect as·quite exceptional; at least I have never met with an example.   The usual combination of left apex-murmurs is that of presystolic with *substitutive* systolic murmur.   Indeed it would be difficult to conceive a perfect first sound occurring where both obstruction and regurgitation exist, and therefore, by inference, disorganization of the valves.

This objection will not apply to the combination of systolic and postdiastolic murmur ("v. systolic and v. diastolic"), because the former may exist at the apex with an imperfect first sound, and the latter at the base with a more or less normal second sound ; or both at the base with sounds more or less normal, the first an accompanying systolic right basic or aortic murmur, and the second a postdiastolic murmur of identical site and origin.

The combinations of organic murmurs most often met with, set down in the order of frequency in which they occur, I believe to be the following :—

1. Right basic-systolic and diastolic (aortic obstruction and reflux).
2. Right basic-systolic and postdiastolic (aortic obstruction and incipient reflux).
3. Left apex-systolic, and right basic-systolic (mitral reflux and aortic obstruction).
4. Left apex-presystolic, and left apex-systolic (mitral obstruction and reflux).
5. Left apex-systolic, and right apex-systolic (mitral reflux and tricuspid reflux).

6. Left apex-presystolic, and right apex-presystolic (mitra
   and tricuspid obstruction).
7. Left apex-postsystolic, and right basic-postdiastolic (in-
   cipient mitral and aortic reflux).
8. Left basic-systolic, and right basic-systolic (pulmonic and
   aortic obstruction).

Non-organic murmurs are divisible into two classes, viz. :
hæmic and *dynamic.* Hæmic murmurs, as the name implies,
are of blood origin, or due either directly or indirectly to an
alteration in the quantity or in the corpuscular constitution of
the blood. They may be located in the heart, the arteries, or
the veins.

These murmurs are sometimes designated as *anæmic* or
spanæmic, according to the view taken of their pathogeny; the
former term implying that the murmur arises from absolute
decrease in the mass of the blood, and the latter, from simple
attenuation or relative reduction in its corpuscular element.

That intra-vascular murmur may be caused by decrease in the
density, irrespectively of the quantity of the blood, seems proven
by the observation of Richardson, that when water in large
quantity is injected into the veins of dogs, a bellows murmur
is developed throughout the entire vascular system.[*]

A similar phenomenon may be witnessed in the human body
in cases of extreme spanæmia from chronic disease of the
primary organ of nutrition; and likewise in chlorosis. In these
cases the volume of the blood would seem to have undergone
no diminution, as testified by the character of the pulse and the
fulness of the superficial vessels. Yet I have repeatedly heard
in such cases a loud bruit de soufflet in the heart and principal
vessels of the body, both arteries and veins.

That absolute loss of blood is competent of itself to produce
murmur, is shown by the occurrence of murmur in the heart and
large arteries after copious hæmorrhage.

Hope[+] narrates a series of experiments performed upon dogs
which likewise go to establish the truth of this doctrine, and
further show in a striking manner what, as being already

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[*] *Medical Times and Gazette,* Lecture, October, 1868

[+] *A Treatise on the Diseases of the Heart and Great Vessels,* third edition, 1839,
p. 99.

well known and universally admitted, it was scarcely necessary to affirm by direct experiment; namely, that increased force and rapidity of the action of the heart intensify the murmur of anæmia, whilst weakening and slowing of it have the opposite effect.

Hope[*] holds "that the murmurs and tremors, as well in the heart as in the arteries, are occasioned by modifications in the motion of the fluid, occasioning increased friction and vibration;" and, to establish this doctrine, he undertakes to prove : 1. "That liquids permeating tubes, do occasion murmurs and tremors." 2. "That in the living subject, modifications in the motion of the blood calculated to elicit murmurs and tremors, do take place under the circumstances in which such murmurs and tremors actually occur." 3. "That the explanation applies equally whatever be the circumstances under which the murmurs and tremors occur." He sustains these propositions by arguments of much force, and by experiments, and concludes that "the physical circumstances usually attending inorganic murmurs in the heart and arteries are, attenuation of the blood; unfilled arteries permitting unusual vibration of their walls and a rippling current; and a certain velocity of the current occasioned by abrupt contractions of the heart."

Andral, whilst accepting unreservedly the doctrine of remote causation of hæmic murmurs by reduced volume or diminution in the corpuscular constituent of the blood, regards as the immediate cause, and dependent upon the preceding, an alleged spasmodic constriction of the orifices at which the murmur occurs.[†] When, however, it is borne in mind that the orifices in question are those of the aorta and pulmonary artery, the immediate boundaries of which are fibrous not muscular in structure, few pathologists will, I apprehend, subscribe this opinion of the illustrious Andral.

Hope,[‡] Beau,[§] and Bellingham,[||] localize the murmur at the orifice of the aorta. This is likewise the opinion of Flint.[¶]

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[*] *Opus citat.*, p. 98.

[†] *Notes au Traité de l'Auscultation de Laennec*, tom. iii., p. 103, Paris, 1837.

[‡] *Opus citat.*

[§] *Traité d'Auscultation*, Paris, 1856.

[||] *Diseases of the Heart*, Dublin, 1853.

[¶] *Diseases of the Heart*, Philadelphia, 1859.

Hughes[*] suggests the orifice of the pulmonary artery as the seat of certain hæmic murmurs, and in the justice of this view I entirely concur. The authors just mentioned agree in regarding blood friction, intensified by attenuation or absolute loss, and accelerated movement of the blood, as the cause of hæmic murmur; and the aortic orifice as its seat when occurring within the heart.

In an article of great literary merit,[†] Dr. Parrot maintains that the seat of cardiac hæmic murmur is the tricuspid orifice, and its cause regurgitation of blood during ventricular systole. I am by no means convinced by the reasoning of M. Parrot; it in many particulars seems inconclusive, and the premises on which it proceeds are erroneous and contrary to clinical experience. For example, I cannot admit that jugular bruit is an invariable accompaniment of cardiac murmur of hæmic origin; nor can I endorse the assertion that such murmur is usually loudest at the inner portion of the left fourth intercostal space, and thence especially traceable upwards and to the right to a certain distance; both of which circumstances would be necessary corollaries of his doctrine as to the cause and site of such murmurs.

Walshe[‡] says, " An intracardiac hæmic murmur of this variety (depending on change in composition of blood) is of moderate or very slight intensity, commonly of medium or low pitch, short or moderately prolonged, of whiffing quality, very easily rendered temporarily harsh by excitement of the heart, and modified in intensity by certain changes of posture.

" This murmur is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta, and of the pulmonary artery, with a force at each proportional to the power of its communicating ventricle; scarcely conducted along the aorta at all; frequently audible, on the contrary, at the second left or pulmonary cartilage; only in exceptional cases audible below the nipple; and never, within my experience, perceptible as far as the left apex."

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[*] *Guy's Hospital Reports,* second series, vol. vii., 1854.

[†] *Archives Générales de Médicine,* Aout, 1866.

[‡] *A Practical Treatise on the Diseases of the Heart and Great Vessels,* third edition, 1862, p 92.

This is a terse and accurate description of cardiac blood-murmurs as usually met with, but conveys no intimation of the author's opinion as to the cause of these murmurs.

Skoda throws doubt on the very existence of blood-murmurs. He says,* " the opinion that murmurs are caused by a particular condition of the blood must be looked upon as hypothetical until it has been shown in what that particular condition of the blood consists. It is not true that a watery state of the blood is a cause of murmurs in the heart; I have many times abstracted very watery blood from patients in whom no murmurs existed.

" It is true that murmurs do occasionally arise in the heart after great loss of blood, and in anæmia, but not so constantly as to justify us in considering the anæmic condition of the blood as the only cause of the murmurs."

The experiments of Richardson, and the general experience of physicians, go to show, as already stated, that spanæmia is of itself quite competent to produce intracardial murmur, and even the high authority of Skoda will be held insufficient to outweigh these considerations.

Again, no well informed physician would venture to maintain that the anæmic condition is the *only* cause of anæmic murmur. It is, no doubt, in the absence of structural disease or debility, the primary and principal cause of such murmur; but, a set of conditions immediately dependent upon it intervenes, and acts as the compound proximate cause of the murmur. To these conditions attention will be presently directed.

At least two factors enter into the causation of hæmic murmurs; viz., 1. *Friction* of the blood corpuscles, (*a*) against one another, and (*b*) against the edges of the opening and the walls of the vessel; and 2. *Vibration* of the heart and walls of the vessels.

Corpuscular friction depends upon ventricular systole, and is directly as the force of latter.

* *A Treatise on Auscultation and Percussion;* translated by Markham, 1853, p. 211.

Chauveau has shown, experimentally,[*] that the rate of circulation in the carotids of a horse was as follows, viz. :

During ventricular systole, 20·4 ; in period immediately succeeding systole and coincident with closure of semilunar valves, 8·6 ; average rate of period succeeding dicrotic impulse and second sound, 5·9 inches per second.

Thus there are three currents of different rates during the period of a single complete action of the heart. After the dicrotic or diastolic impulse the rate grows gradually slower, till just before the repetition of the impulse (*i.e*, during the momentum of auricular systole), when it is almost *nil*.

He also found that as the small arteries are approached the systolic impulse rapidly diminishes, and the dicrotic (diastolic) impulse becomes very feeble or is entirely abolished. The *constant* flow, however, is very much increased in rapidity. Thus it would seem that by the elasticity of the large arteries the impulse, both systolic and dicrotic, is gradually converted into a continuous force, under which the flow in the smaller vessels becomes constant, and by an equal distribution of that force, also more rapid ; that the current in the large arteries is intermittent ; in those of medium size remittent ; and in the very small arteries nearly constant.[†]

Increase of rate no less than of force of current has the effect of intensifying friction ; but, as both force and rate of circulation are at their maximum during ventricular systole, it is quite intelligible that a murmur whether organic or non-organic, depending upon blood-friction, should be systolic in rhythm ; and furthermore as, other circumstances being equal, friction must be greatest where the passage is narrowest, the seat of murmur of either kind will be that of the most constricted portion of the vessel. Now, even in the healthy state, the narrowest portion of the aorta and pulmonary artery is the orifice of both vessels ; hence the occurrence of hæmic murmur by preference in these situations.

It is worthy of remark, as affording further evidence in support of this view, that whilst hæmic systolic aortic murmur may or

[*] *Nouvelles Recherches Experimentales sur les Mouvements et les Bruits Normaux du Cœur envisagés au point de vue de la Physiologie Médicale*, Paris, 1856

[†] *Physiology of Man*, by A. Flint, vol. i., 1866.

may not, according to the force and efficacy of its several contributory causes, be accompanied by murmur in the arteries, the latter *never* occurs unaccompanied by murmur at the orifice of the aorta, except where caused by extrinsic pressure upon the vessel, morbid narrowing of its calibre, or roughness of its interior.

Vibration of the heart or vessels is due in part to friction; but likewise, in some degree, to the existence of an eddy or ripple in the passing current. In organic disease this is, in many instances, sufficiently pronounced to be perceptible to the coarser sense of touch; but in cases of non-organic murmur due to modified constitution of the blood, vibration is never detectable by the hand as fremitus, though manifest to the finer sense of hearing.

Chauveau has shown, experimentally, that when a liquid current is hurried past a constriction in its passage into a wider space beyond, a ripple or perturbation in the current is created, giving rise to vibration and murmur.

Corrigan, in 1829, clearly established this by experiments conducted upon the intestine of an animal. He says :* " While the intestine was tense no sound, or a murmur exceedingly indistinct, was heard; but any part being constricted so as to produce an alteration in the motion of the fluid, a very loud bruit de soufflet immediately became evident." I have already criticised this passage (p. 172) on the ground of its containing a denial of friction-murmur from water-currents passing under strong pressure through tubes, whether solid or not, and independently of constriction. I may add that I cannot accept the conclusion of the author that, in experiments of the kind described by him, murmur is audible only on the *distal* side of the constriction; because the influence of friction as a factor, would be thereby in effect excluded. I have carefully repeated Corrigan's experiments, using a human intestine under a water-pressure of several pounds to the square inch, sufficient ultimately to rupture the intestine, and have satisfied myself, and my assistants, of the existence of bruit on the *proximal* side of the constriction produced by means of a strong cord. Still,

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* *Lancet*, April 11th, 1829.

Corrigan has established the main fact of the existence of ripple, and consequent vibration and murmur, beyond the seat of constriction.

Friction, whether inter-corpuscular or vaso-corpuscular, in a degree adequate to the production of murmur in blood impoverished as to its solids, or diminished as to its absolute volume, is due mainly to atony or relaxation of the vascular walls, and low arterial tension consequent upon malnutrition of the vaso-motor nerve centres.

Vibration is, I believe, traceable to the same cause. I am glad to find that in this opinion I am fortified by the authority of M. Marey, who says: "The murmurs which anæmia, chlorosis, and fever produce at the level of the orifice of the aorta, are due to lowering of arterial tension, and the greater rapidity with which ventricular systole is accomplished."*

In this state of the arteries, and owing to the constant and rapid oscillation of their walls, the blood does not move in an equable current, but is thrown into a series of ripples or eddies by which collision and friction are multiplied. Should constriction or other obstruction to the onward flow of the current present itself, vibration, as shown by Corrigan and Chauveau, will be intensified, and the murmur proportionately louder.

There can be no doubt that, as long since urged by Stokes,† many organic murmurs are reinforced, or even in greatest part constituted, by morbid sound arising from the friction and vibration of impoverished blood in transit through rough or constricted passages; and that the alleged cure of organic disease of the heart, and the actual improvement of the patient's health which so often follows tonic, and especially chalybeate treatment persistently continued, are to be attributed to the improved condition of the blood, and the consequent cessation of murmur *quoad* anæmia.

The *diagnosis* of hæmic cardiac murmurs has reference mainly to the distinction between such murmurs and those of organic origin; and, in most instances, may be confidently made from a careful consideration of the actual physical signs and the symp-

* *Arch. Général de Médecine,* par le Dr. Parrot, Aout, 1866.
† *Diseases of the Heart and Aorta,* 1854.

toms exhibited by the patient. In the few instances where this is not possible, a full consideration of the antecedent history of the case, and further observation of the effect of treatment, which in doubtful cases should always proceed on the assumption of the murmur being of hæmic origin, will remove all difficulty. Cases of this doubtful nature are usually of mixed character, being due to anæmia and to structural alteration in varying relative proportions; and the degree in which each of these causes operates can be determined only by treatment. If, in any given case, abatement without cessation of the murmur, and improvement in the general condition of the patient result from the persistent use of chalybeates and of regimen adapted to the amelioration of the blood, not only may the diagnosis of a mixed case be confidently made, but the *degree* in which the humoral and the structural element had respectively operated, may be likewise satisfactorily determined by a comparison of the previous with the actual signs and symptoms.

Cardiac murmurs of hæmic origin are invariable basic, they are loudest at midsternum or in the anatomical site of the orifices of the aorta and pulmonary artery; with this point as centre they have a diffusion-area of three to four inches in diameter, according to the intensity of the murmur and the conducting qualities of the chest wall; but they never exhibit a definite line of propagation, as is the case with organic murmurs in this situation.

Not unfrequently, but only in aggravated examples, murmur likewise exists at some point of the ascending and transverse aorta, and in the carotid and subclavian arteries, but never, as far as I have observed, in unbroken continuity throughout these vessels; a feature in which they differ from organic murmurs at the orifice of the aorta. The latter are, moreover, not infrequently audible at the apex, though with diminished intensity; the former never.

Finally, in the recumbent posture feeble hæmic murmurs are in many instances inaudible, but are promptly developed when the patient sits up; they are usually soft and blowing in quality but subject to considerable variation in this respect, assuming occasionally a sharp or sawing character; whereas, organic aortic

murmur is never entirely suspended, save in extreme debility of the patient, and undergoes, with change in the posture of the body, an alteration in degree of loudness but not in quality of sound.

In the principal arteries of the body, down to those of the third order, murmur may be developed in certain pathological conditions.

Bouillaud maintains that in a state of health murmur is produced in these vessels by the shock and friction of the blood against the vascular walls at each stroke of the left ventricle.*

According to him, arterial murmur varies with the strength of ventricular contraction, liquidity of the blood, and the thickness, tension, and internal smoothness of the arteries. Thus the physiological murmur passes by imperceptible gradations into that which is recognized as pathological, and named by him *bruit de soufflet ordinaire et intermittent.*

I cannot admit the existence of physiological arterial murmur, although *a priori* I should have expected it, bearing in mind the occurrence of murmur in metallic or elastic tubes through which water passes under strong pressure. As a matter of fact, however, I have failed to discover this phenomenon in the arteries of the healthy body;† and if speculation were not prohibited in a science of experiment, I would venture to suggest, whether its absence may not be due to the layer of white corpuscles by which normally the vessels are lined, and by which friction of the passing current against the vascular wall may possibly be obviated. Arterial murmur in the pathological sense may arise, according to Bouillaud, from, 1. Pressure on the artery in which it exists; and of this he offers as an example the so-called *bruit placentaire* which he attributes to pressure of the gravid uterus upon the iliac arteries; 2. Aneurism; 3. Roughening of internal surface of artery by calcareous or cartilaginous plates with or without narrowing; 4. Passage

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* *Traité Clinique des Maladies du Cœur,* tom. i., 1835.

† If the noise produced by sudden tension of the vessel, and friction of it against the end of the stethoscope during expansion be excluded, there is nothing to present even the semblance of murmur.

of blood from an artery into a vein (in this instance, however, the murmur is venous not arterial) ; 5. Great agitation of the arterial system as in anæmia and chlorosis.

In addition to intermittent arterial murmur, synchronous with ventricular systole and arterial diastole, which is the ordinary form in which this phenomenon is presented, Bouillaud admits three others, namely, *bruit de soufflet continu, ou a double courant; bruit ou soufflement de diable;* and *sifflement modulé, ou chant des artères.*

Bruit de soufflet continu, he describes as consisting of two murmurs, seemingly continuous, the first of which is very loud like that of a forge bellows, and succeeded immediately by another much less loud; sometimes it resembles the cooing of a turtle dove, sometimes the whistling of the wind through a chink or key-hole. It occasionally passes by a gradual ascent into the *bruit de diable,* and may be always made to do so by regulated pressure.

Bruit de diable, so named from its resemblance to the whizzing noise made by the "diable," which is a French toy played by whipping, is regarded by Bouillaud as arterial in origin, and most frequently heard in the carotid and subclavian arteries; usually on one side only and more frequently the left than the right; but according to Laennec it is more common on the right side, and in this opinion I concur. Even when audible on both sides it is louder on one side than on the other. It is subject to capricious and irregular intermittences which are at present inexplicable. Gentle pressure with the stethoscope sometimes enfeebles it, sometimes converts it into a growl. It is rendered louder by turning the head to the side under examination, and enfeebled or suspended by lifting the larynx from the arteries. Prolonged effort of the person examined, and pressure on the vessel above the point auscultated, promptly suppress it.

The general accuracy of this graphic sketch of bruit de diable by Bouillaud I readily admit; but, with the most recent writers on this subject, I regard the phenomenon as of venous origin, and as having its seat in the jugular veins. Indeed, it is now very generally named "jugular murmur," or "venous hum."

The fact, admitted by Bouillaud, that gentle pressure *above* the point examined will suffice to suppress the murmur, is *prima facie* evidence against his doctrine; and, still further, the now universally admitted fact that this phenomenon is all but confined to the right side of the neck, points directly to the superior cava and its tributaries as being its seat. Finally, its continuous though modulated character may be regarded as confirmatory of this view.

The musical arterial modulation described by Laennec, as consisting of " musical tones as if the artery were become a vibrating string from which two or three notes were drawn out in succession by advancing and drawing back the finger upon it,"* distinguished by Bouillaud as *sifflement modulé ou chant des artères*, and compared by him to the buzzing of a fly, is peculiar to delicate, nervous, and anæmic subjects, as correctly stated by Bouillaud. In such subjects, usually females, I have occasionally heard it in the arch of the aorta and its primary branches, but not elsewhere; it consists of a whistling, squeaking note, variable in pitch and quality, and generally inaudible when the body is recumbent and the nervous system in a state of functional quiescence, but promptly brought out by mental excitement or sudden assumption of the erect posture. In one notable case the arteries were remarkably tortuous, and the patient was liable to sudden and unaccountable flushings of the face. This musical note ascends with the ventricular systole, and usually, but not invariably, descends with the recoil of the arteries; its precise mode of production is not known, but as to cause, it is unquestionably a nervous phenomenon.

In the remarkable case above alluded to, it was noticeable that the patient's face became suddenly flushed on my appearing at her bed-side during the morning visit, and coincidently the murmur was unusually loud; and on visiting her again before leaving the ward I have repeatedly found the face pale, the pulse tranquil, and the murmur all but, or even quite, inaudible. Whether arterial lesion, other than the tortuous condition already alluded to, existed in this case, I cannot say; and it is probable that, as palpitation and marked acceleration of pulse always

* Forbes' translation, p. 557.

preceded increase of the murmur, the nervous influence accessory to it operated not directly upon the vessels, but through the heart. It is not difficult to conceive that the heart propelling with increase force and rapidity successive volumes of blood into the primary arterial trunks, would, if these were flexuous, be competent to develope in them a murmur of very high pitch, synchronous with the contraction of the ventricles, and occasionally also continued into the systole of the artery.

In several instances I have heard the simple arterial souffle in the arteries of the neck and shoulder, when no murmur whatever existed in the heart or aorta. In the carotids I have heard such a murmur in the person of a nervous female, with a weak and dilated heart, a month after confinement; it existed independently of pressure, which, however, rendered it proportionately louder.

I confess I am unable to account for strictly localized and transient carotid murmur in this and many similar cases, in which no palpitation of the heart, and presumably no arterial disease existed; and certainly no enlargement of the thyroid, or other cervical tumor. Is it to be regarded as the result of local arterial inhibition through the agency of the great splanchnic, or vaso-inhibitory system of nerves?

With the subclavian arteries, however, the case is different. In these vessels I have in numerous instances heard bellows murmur in two different connexions, and of widely different import; namely (a) in cases of disproportionate development of the subclavian muscle, as in artificers, such as blacksmiths, sawyers, etc., who use their arms much in the pursuit of their calling; and (b) in the subjects of primary tubercular deposition in the extreme apex of the corresponding lung.

Doctor Richardson avers* that he has never examined the chest of a carpenter, cabinetmaker, or sawyer, without finding subclavian murmur. He states, and very correctly, that where it exists it is always intensified by the subject under examination holding the corresponding arm horizontally or at right angles with the side, and that in many cases it is audible only under these latter circumstances. This he accounts for by

* *Medical Times and Gazette,* 1870.

assuming compression of the artery between the subclavius muscle and the first rib.

I have repeatedly met with subclavian murmur in persons of the calling mentioned by Dr. Richardson; but in many, likewise, I have failed to find it. I have, moreover, repeatedly heard murmur in this vessel in persons whose subclavian muscles could not have been assumed to have undergone excessive development by reason of their avocations, and yet in whose lungs there was no evidence of tubercular or other solidification.

In all these cases, however, the murmur was either brought out or intensified when the patient held out his arm, horizontally, for a few moments, by the action of the muscles of his shoulder. I am therefore of opinion, that Dr. Richardson's theory is correct *quoad* his explanation of the abstract phenomenon. I believe that murmur is neither audible in *all* who pursue the handicrafts which he has named, and that it is not limited to such exclusively ; although undoubtedly of more frequent occurrence in the trades which he has mentioned than in any others. I am fully convinced of murmur due to this cause. In regard to the absolute proportion of *all* cases in which subclavian murmur occurs, and the influence of sex and age in its production, Dr. Richardson states* that he has met with it in 51 cases out of 2,000 : *i.e.*, in 2·55 per cent. Of these 2,000 patients, 1,011 were females, and 989 males; yet the murmur existed in 48 of the males, and in only 3 of the females; the youngest patient presenting this murmur was aged 18, and the oldest 74 years. It would thence appear that subclavian murmur is of much more frequent occurrence in the male than in the female sex, and that it may be present at any period of life from puberty to old age.†

Subclavian murmur in persons of phthisical proclivity is of much greater diagnostic and practical importance. In these it exists usually on one side, and only at the acme of inspiration. During this portion of the respiratory act it is remarkably loud, and is repeated once, twice, or oftener, according to (*a*) the dura-

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* *Loco citat.*

† Dr. Leared (*Medical Times and Gazette*, October 31st, 1868,) prefers to attribute arterial murmur, generally, to local arterial contraction from reflux irritation.

tion of the inspiratory effort, and (b) the rate of pulsation of the heart, corresponding exclusively with ventricular systole. Subclavian murmur having the connexion and characters now mentioned, viz., co-existence with certain presumptive symptoms, with or without other physical signs of incipient pulmonary phthisis, and being audible at one side, at the acme of inspiration only, irrespectively of the position of the corresponding arm, I regard as strong evidence of solidification of the apex of the lung. I am limited for confirmation of this view, however, to observation of the subsequent progress of only a few such cases, and have not had the advantage of a *post mortem* examination of the body in any. Nor is it difficult to afford a rational explanation of the phenomenon. At the acme of inspiration the apex of the lung glides forwards beneath the arch formed by the subclavian artery in its first and second stages, brushing its anterior surface forcibly against that vessel in its ascent.

If in this situation, namely, where the imprint of the vessel in the shape of a semicircular indentation on the anterior surface of the apex may be observed in the healthy lung, the pulmonary structure has become solid, it is conceivable that a murmur in the vessel may result from pressure made upon it by the solid portion of the lung, at the moment when the parts are brought into mutual contact, by ascent of the apex in inspiration.

Venous murmur is a phenomenon of considerable speculative interest; it has afforded, and still continues to afford, subject matter for the most ingenious and diverse speculations. It was first observed by Laennec, who compared it, not inaptly, to the roaring of the sea heard at a distance; and, still more appropriately, to the continuous but undulating sound heard on applying a spiral sea-shell to the ear. Laennec, however, located this phenomenon in the carotid arteries, alleging that it was occasionally audible likewise in the heart. This opinion was adopted and advocated, with much force and ingenuity, by Andral, Bouillaud, Corrigan, Laharpe, Vernois, Marey, and Beau.

In 1837, Dr. Ogier Ward,* of Birmingham, propounded the doctrine of the venous origin of the continuous cervical murmur,

* *Medical Gazette,* vol. xx., p. 7.

up to that time regarded as having its seat in the arteries of the neck

Hope adopted this opinion, which was subsequently professed by Hughes, Bellingham, Walshe, and Stokes. In France, Aran, Moneret, Hardy and Béhier, Chauveau, Barth and Roger, and Parrot, advocate the same view, which is now all but universally held by physicians.

Laennec regarded this murmur as a purely nervous phenomenon, and due immediately to spasmodic contraction of the arteries.

Bouillaud considers that whilst several causes contribute to its production, the most potent is friction of the blood against the walls of the vessel when that fluid has undergone changes of elementary constitution; and he surmises that the proximity of the heart, larynx, and trachea may have some share in its production. He admits, however, his inability to assign to each of these alleged causes its due value, or to declare that other and still unknown causes may not co-operate to produce this concrete phenomenon.

Andral, with much reserve, held the opinion that the murmur was due to the friction of an impoverished blood, projected with inadequate force by a weakened left ventricle into arteries partially contracted.

Beau holds to an alleged disproportion between the volume of the blood and the containing arteries, and consequent friction.

Bellingham denied the existence of venous murmur, save when pressure is made with the stethoscope upon the vessel, and its walls are thereby made tense.

Walshe, likewise, considers pressure upon the vein indispensable to the production of murmur within it; although, with Hamerngk and Skoda, he holds that two predisposing conditions must exist, namely, a state of permanent dilatation of the vein, and a reduced volume of blood circulating through it.* The former of these conditions is presented by the internal jugular, only at the point where it debouches into the innominate vein. Here the walls of the vessel are kept permanently on the stretch,

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* In the fourth edition of his book (1873), Dr Walshe does not reaffirm this theory. Indeed, with the single exception of spanæmia, he declines to associate the phenomenon with any definite condition of the veins or their contents

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and prevented from collapsing, by the deep cervical fascia, which enfolds the vein, and is attached to the fixed points of the clavicle and sternum below, and to the omohyoid, the sterno-hyoid, and sternothyroid muscles on either side.

The reduced stream of blood descending from the vessel above, into this dilated and patulous portion of it, assumes a spiral course; and, by the exaggerated friction and vibration of the tense walls of the vessel thereby produced, becomes a cause of murmur and fremitus.

This is an exceedingly ingenious theory, but at least two fatal objections may be urged against it; namely, the *necessary* assumption of a reduced volume of blood, and the *necessary* consequence of frémissement. It is notorious that venous murmur may be associated with a simply spanæmic state of the blood, irrespectively of decrease of its mass ; and no less so, that in a large proportion of the cases in which it is heard no fremitus can be detected.

Marey,* who adheres to the doctrine of the arterial origin of the murmur in question, regards anæmia as a remote or indirect cause of the murmur by lowering arterial tension; in consequence of which the capillaries permit a more ready outflow. Hence, when pressure is made with the stethoscope on one of the large arteries, the portion of the vessel beyond the point subjected to pressure is rapidly emptied of blood through the capillaries, or tends to be; its walls become relaxed, vibrate, and produce murmur.

It is not necessary to combat this doctrine, which is based upon an erroneous view as to the seat of the murmur; otherwise it would be sufficient for this purpose to remark, that the murmur is much louder on the cardiac than on the distal side of the point of pressure.

M. Vernois† thought the murmur arose from friction of the blood against the folds which were formed in the walls of the arteries, consequent upon reduction in volume of the contained blood.

M. De La Harpe, considering only the density of the blood,

* *Physiologie Médicale de la Circulation du Sang*, p. 476, Paris, 1863.

† *Etudes Physiologiques et Clin. des Bruits des Artères.* (Theses de Paris, 1837, No. cccclxxviii.)

compared the arterial current to that of a column of air in a pipe, yielding, like the latter, sonorous vibrations.

M. Aran,[*] regarding continuous and musical murmurs as of venous origin, attributed them to increased rapidity of circulation, and to friction, both intrinsic and against the walls of the vessel, consequent upon impoverishment of the blood. Such was likewise the opinion of Barth and Roger.

M. Chauveau,[†] holding these murmurs to be venous, attributed them to the vibration of a central current, or "fluid vein," which is formed in the roots of the venæ innominatæ, owing to the inability of this portion of the vessels to accommodate itself to the reduced volume of the blood (see page 183).

M. Heynsius,[‡] considers vascular murmurs, in general, due to a peculiar movement of blood in a dilated portion of the vessel, and beyond a constriction. He holds that local contraction in the calibre of a vessel, must create eddy and murmur in the dilated portion in advance of the constriction.

M. le Dr. Parrot[§] concludes from his observations that venous murmurs in the neck, both intermittent and continuous, are due to vibration of the blood-corpuscles produced by the passage of a current, afflux or reflux, through the embouchure of the internal jugular vein.

When the murmur is intermittent, it is synchronous with auricular contraction and jugular pulsation, and is caused, according to Parrot, by the passage of a reflux current, under the pressure of auricular systole, from the temporarily distended vena cava into the relaxed jugular ; the orifice of debouchment of the latter vessel being now incompletely closed by its valves.

The continuous murmur, or bruit de diable, is composed of two elements: namely, that just described, and another, less loud, which immediately succeeds it, and which is itself caused by the onward flow of blood from the jugular, now distended by the

[*] *Opus citat.*

[†] "Etudes pratiques sur les Murmures Vasculaires ou Bruits de Souffle, et sur leur valeur Séméiologique." (*Gazette Médicale de Paris*, 1858, p. 247, et suiv.)

[‡] *Journal de la Physiologie*, tom. iii., p. 722.

[§] *Archives Générales de Médecine*, 1867, tom. i., p. 649, *et suiv.* To the learned author of the last quoted article I am indebted for many of the preceding references, and for much valuable information on this subject.

preceding auricular systole, into the cava, relaxed coincidently with the commencement of auricular diastole.

It would be difficult to explain why, in this view of its causation, venous murmur should not exist in every case of engorgement of the right chambers of the heart. Yet, such is notoriously not the case; whilst, on the contrary, venous murmur is usually unassociated with venous engorgement. Besides, careful attention will show that the murmur is one of afflux exclusively. Neither can I assent to the doctrine of M. Parrot, that few persons, even in good health, are free from venous murmur; in fact, that its presence is the rule, and its absence the exception.

Hope has taken a more correct view of the conditions and cause of venous murmur, regarding it as a symptom of anæmia; under which term he includes not only diminution of volume, but likewise attenuation of the blood. According to him, it is immediately due to the twofold cause, of ripple of the current, and vibration of the walls of the vessel; and few persons exhibiting any degree of anæmia are free from it. He does not, however, attempt to explain the connexion of these proximate causes of murmur with a simply hydræmic condition.

Hope very correctly states that the ordinary venous murmur is a continuous bruit de soufflet, but liable to augmentations due to the *saccade* of the adjacent artery, and to certain modifications of tone from pressure upon the internal jugular vein, in which the murmur is most frequently located. The external jugular, and, indeed, all the large and medium-sized veins of the body may, however, be the seat of murmur in aggravated anæmia.

The rhythmical augmentations of the murmur very generally met with, coinciding as they do with ventricular systole, were regarded by Bouillaud as constituting the second element of a duplex arterial murmur. By gentle pressure upon the vein, the murmur may be so altered in quality as to resemble the noise made by a child's humming-top; this is the "venous hum" of Hope; a designation certainly more intelligible, at least outside France, than the synonyme "bruit de diable." Occasionally, and to appearance capriciously, the murmur undergoes a still further

elevation of pitch, and assumes a musical character ; aptly compared by Hope to a delicate whistle produced by the lips, the noise made by the wind passing through a key-hole or crevice, the singing of a kettle, or the song of a mosquito, in different examples. In all these cases, I have no doubt that the variation of murmur is due to alteration in the rate of circulation, of vascular pressure, or of tension of the walls of the vessel, separately or conjointly ; and consequent upon an insensible movement of the body, contraction of adjacent muscles, tension of the integument, or vaso-motor influence operating on the heart or directly upon the vein.

Respiration exercises a marked influence upon jugular murmur. As stated by Hope, it is augmented by a full inspiration, when it assumes a *rushing* character ; and by a prolonged or forced expiration, it is all but, or even entirely suspended. Gentle pressure above the point auscultated renders the murmur louder ; and strong pressure, above or below this point, has the effect of altogether suppressing it.

I agree with Laennec and Hope in the opinion that venous murmur is always louder on the right than on the left side, the authority of Bouillaud to the contrary notwithstanding ; but, I cannot subscribe the statement of Hope, that in *all* cases where it is audible on the right, it is likewise, though in a less degree, present on the left side. I have repeatedly noted its absence on the left side, when distinctly audible, though not loud, on the right.

Niemeyer* says the bruit de diable is louder on the right than on the left side, and that it ceases when the patient assumes the recumbent posture and makes a full inspiration. He adopts the hypothesis of Hamerngk and Skoda, as to the mode of its production ; viz., that it is due to a twofold cause : permanent expansion of the internal jugular at its termination in the innominate vein, and a vortigenous movement of the diminished blood-current in passing through this portion of the vessel, and consequent vibration of its tense walls. The objections already urged against this theory, are not in any degree

* *A Text-Book of Practical Medicine*, translated by Humphreys and Hackley, New York, 1869, vol. ii., p 723.

lessened by the sanction it receives from the high authority of Niemeyer; it is no less characteristic of simple spanæmia when the blood has suffered no diminution of volume, than it is of the condition induced by absolute loss of blood. Nor can I assent to the statement of Niemeyer, that venous murmur may be induced in persons free from any abnormity of the blood, by causing them to rotate the head towards the opposite side; when, as he believes, the pressure made upon the vein by the omohyoid muscle becomes a cause of murmur. I have never heard genuine venous murmur in healthy persons under the circumstances just mentioned, nor as a consequence of simple pressure upon the vein, however made.

Bruit de diable, then, I regard as a sign of absolute reduction of volume, or of attenuation of the blood. It is frequently absent on the left side, and, when present on both sides, is always louder on the right. It is intensified when the patient changes from the recumbent to the sitting posture, and, for a moment, ceases to be audible on resumption of the former of these postures; but, after a brief interval, it may be again heard, though with diminished intensity. It is louder during inspiration than during expiration, and by holding the breath, is entirely suspended. Light pressure upon the jugular above the clavicle, whether made with the stethoscope, the finger, or through the muscles by rotation of the head, intensifies the murmur, whilst stronger pressure entirely stops it. It gradually ceases under appropriate treatment directed to the faulty condition of the blood, and is, on the contrary, made more distinct by causes which aggravate that condition. It is remarkably variable, and apparently capricious in regard to pitch, tone, and absolute loudness; but these variations may in every instance be traced to difference in the rate or force of the heart's action, or to variation of tension in the walls of the vein itself, under vaso-motor or vaso-inhibitory influence.

Venous murmur, though most frequently heard in the jugulars, is by no means confined to these vessels; it is present in aneurismal varix, and in the placental and uterine veins in the latter months of pregnancy. I have likewise repeatedly heard it in the sinuses of large myomatous tumors of the uterus, and once,

in the dilated veins ramifying upon the surface of a large osteo-sarcomatous tumor of the arm of a young anæmic man, subsequently operated on, successfully, by my friend and colleague, Mr. Tyrrell. Indeed, in anæmic subjects, gentle pressure on any of the large veins will suffice to elicit this murmur.

As to the precise mode of production of these murmurs, it may be positively affirmed that they are, like arterial murmurs, due to the multiple cause of mutual collision of the blood-corpuscles, friction of the blood against the walls of the vein, and vibration of the latter. As to why these causes operate with effect in the anæmic or spanæmic condition exclusively, it were vain to attempt an explanation in the present state of knowledge; as the inadequate and inconclusive character of the conjectures, previously referred to, sufficiently testifies.

Dynamic murmurs are exclusively cardiac, and located at the auriculo-ventricular orifices; they are presumably attributable to irregular contraction of the ventricles, and imply the absence of permanent dilatation or valvular disease. All writers agree in regarding them as exclusively regurgitant, but two theories are held as to the precise mode of their occurrence; namely, that they are the result, upon the one hand, of disorderly or excessive contraction of the papillary muscles; or, upon the other, of asynchronous or imperfect contraction of these, or of a limited portion of the ventricular wall. The former of these theories has been applied in an especial manner to explain the evanescent murmur of chorea, whilst the latter has been adduced to account for the murmurs so frequently met with in adynamic fever, and in nervous exhaustion.

Laennec was probably the first who directed attention to murmurs of this class. He says,* "I can state with certainty that the bellows sound of the heart is very often met with when the organ is perfectly healthy." This statement applies, no doubt, equally to anæmic and dynamic cardiac murmurs, which were not distinguished by Laennec, and were alike attributed by him to spasm of the heart.

Dr. Todd, who, so early as 1843, insisted on the connexion between chorea and rheumatism, reiterated this opinion with

* Forbes' translation, p. 559.

much emphasis in his Lumleian Lecture in 1849.* In this lecture he dwells upon the frequent occurrence of mitral systolic murmur in chorea, and regards it as due to organic valvular lesion, arising from "insidious endocarditis" of rheumatic origin. Thus, he considers rheumatism to be the starting point of chorea, but mitral organic lesion to be a necessary link in the chain.

It is not possible, however, to accept this as a satisfactory solution of the difficulty ; because, as Dr. Todd himself remarks, the natural tendency of chorea is to get well. He adds, " it is rare to meet with any symptom of heart affection either preceding or accompanying the chorea;" and this, notwithstanding the admittedly large per-centage of choreics in which systolic apex-murmur exists.

It would seem that the causal connexion of rheumatism with chorea, was recognized at Guy's Hospital so early as 1802.† In 1837, Dr. Bright advocated a close and immediate connexion between pericarditis and chorea. In 1839, he gave examples of various forms of muscular spasm, including chorea, dependent upon pericarditis, and immediately due, as he believed, to peripheral irritation of the phrenic nerves.‡

Dr. Bright, it is true, did not advert to endocardial inflammation or murmur in connexion with chorea, although in one of his cases endocarditis actually existed; but his observations are important, collaterally, as a contribution to the theory of choreic mitral murmur.

In 1850, Dr. Botrel declared§ that chorea was only a special manifestation of rheumatism ; and in the following year (1851), Dr. G. Sée went further, holding that in nearly every case of chorea rheumatic symptoms were manifested; in many, however, only in the form of pain.

Trousseau|| only contends for " a law of coincidence," as between rheumatism and chorea ; holding that while either may take precedence, rheumatism generally does so, and that chorea actually complicates rheumatism in the proportion of one-third

* *Medical Gazette*, vol. viii., 1849, p. 663.

† Bright, in *Med. Chirurg. Transactions*, vol. xxii., 1839.

‡ *Loco citat.*

§ *Chorea considered as a Rheumatic Affection*, Special Thesis, quoted by Trousseau.

|| *Lectures on Clinical Medicine*, New Sydenham Society, 1868, vol. i., p. 395.

of the cases. Under the head of rheumatism, however, he admits without reserve, endocarditis; assuming, with Bouillaud, that every case of endocarditis is of rheumatic origin. So firmly rooted is this opinion, that, forced to admit the occasional occurrence of endocarditis in the course of scarlatina, he would save his doctrine by alleging that rheumatic arthritis is a common accompaniment of scarlatina. I have repeatedly observed chorea unconnected by history or symptoms with rheumatism, scarlatina, or valvular murmur. That articular pains are not of frequent, or indeed of more than exceptional occurrence in scarlatina, and then only in the stage of desquamation and as a consequence of indirect exposure of the limbs to cold, is my firm belief.

Doctor James Begbie* gives three family histories illustrative of the association of rheumatism, acute inflammation of the pericardium or endocardium, and chorea, either in the same person, or by hereditary transmission. These cases are not, however, conclusive in support of a necessary connexion or kindred association of these affections. In some of the examples neither rheumatism nor cardiac disease existed; and chorea was directly traceable to fright or imitation, both very common causes of the affection. Dr. Begbie is of opinion that the connexion between rheumatism and chorea is to be explained by reference to the morbid condition of the blood in the former of these diseases.

Babington thinks that chorea, following affections of the heart or pericardium, is due to reflex irritation through the cardiac nerves and ganglia; but he admits the possibility of inflammation of the spinal meninges.†

Doctor Burrows‡ also explains the connexion by reflex irritation of the phrenic and pneumogastric nerves.

Sir T. Watson surmises that the connexion between chorea and cardiac disease may be due to their common dependence upon the rheumatic diathesis. In virtue of its tendency to invade fibrous structures, rheumatism may give rise, at the same time, to acute inflammation of the membranes of the spinal cord and of

* *Contributions to Practical Medicine*, 1862, p. 68.
† *Guy's Hospital Reports*, vol. vi., p. 418.
‡ *On Disorders of Cerebral Circulation*, section vii.

those of the heart; and thus become the cause of chorea by mechanical irritation of the roots of the spinal nerves through inflammatory exudation and thickening of their sheaths; and also of pericarditis or endocarditis, or of both. The gravamen of the disease may fall on the spinal cord or the heart, separately or consecutively. He suggests, also, that the cardiac lesion may be an excentric cause of the spasmodic disorder of the muscles.[*]

In the last edition of his work, the distinguished and veteran author modifies his former opinion; abandoning it, in effect, in favour of Kirkes' doctrine, in support of which he dwells upon the preponderance of *mitral* over other cardiac murmurs, as proving the existence of organic lesion of the valves. He adds: " I, for one, accept the conclusion that has been drawn from them (the facts and reasonings which connect chorea with valvular disease on the left side of the heart), namely, that the chorea is often, not always, the ultimate result of the injection of molecular particles of fibrine into some of the minute arteries or capillaries of the nervous tissue."[†]

Doctor Kirkes denies that the mitral reflux murmur of chorea may be due to choreic or irregular action of the papillary muscles, on the ground that "there is no good proof that involuntary muscular organs participate in the choreic disorder." There is much force in this objection; but, the admitted co-existence of rhythmical action of the substance of the heart, with this alleged tetanic and irregular contraction of the papillary muscles, which, as proved by dissection, are directly continuous with the fibres of the ventricular walls, constitutes a still stronger objection. We should be forced to admit, on the assumption of this theory, that whilst one and the greater portion of the length of certain muscular fibres contract with perfect order and regularity, the remaining and smaller portion of the same fibres act spasmodically, and out of harmony with the former. But no physiologist, bearing in mind the unity of nerve-centres, and the community of nerve-distribution enjoyed by both portions of the same fibres, could admit such a doctrine.

* *Lectures*, vol. i., p. 664.

† *The Principles and Practice of Physic*, fourth edition, 1871, vol. i., p. 674.

Doctor Kirkes urges the opinion,* and defends it with much ability, that chorea is directly due to organic disease of the mitral valve. He believes that rheumatism has a causal relationship to chorea, only so far as it contributes to the production of endocarditis on the left side of the heart; and, in confirmation of this view, dwells upon the facts that chorea shows itself only towards the close of the rheumatic attack, or even weeks or months after it; that there has been no evidence of a rheumatic tendency in at least one half of the fatal cases of chorea which he has witnessed; and that valvular disease of the heart may arise, irrespectively of rheumatism, from pyæmia, the exanthemata, pregnancy, and other causes. In chorea, moreover, endocardial inflammation engaging the valves of the left side, and giving rise to granular vegetations upon them, is a very frequent, if not an invariable, accompaniment of the nervous affection. He admits, however, that in rare instances chorea may occur independently of endocarditis; but he holds that in all rheumatic cases endocarditis is a necessary link, the cause being twofold; viz., contamination of the blood by the rheumatic or inflammatory products, and obstruction of the capillaries by disintegrated fibrinous deposit.

One statement of Dr. Kirkes I deem it necessary to give in his own words, for the purpose of recording my dissent from it. He says: " One important point in connexion with these choreic murmurs requires to be especially noted, namely, that absence of murmur is no proof of the absence of even serious organic disease of the valves of the heart. I have repeatedly observed cases in which the most careful examination failed to detect a murmur, even up to the last day of life, yet in which after death there were unmistakable signs of recent acute mischief about the valves;" and, by way of explanation, he adds, " The thickening, swelling, and other changes in the mitral valve, including fibrinous depositions, were, in the cases to which I allude, observed principally, if not exclusively, on the auricular surface of the valve, above the free margin, which was thus uninterfered with in its power of closing and preventing regurgitation."

* *Medical Times and Gazette*, 1850 and 1863.

The accuracy of the observation upon which the preceding statement is made, I have no right to question. I can only express my astonishment, and declare that it is utterly at variance with all I have learned on this subject.

I have never witnessed a case in which *post mortem* examination showed the existence of "recent acute mischief," such as " thickening, swelling, and other changes in the mitral valve," much less "fibrinous depositions," upon it, in which at least faint murmur, or characteristic softening and prolongation of the first sound, had not been present at some period of the patient's last illness. Absence of this physical sign on the last day of life, or even for several days preceding death, owing, no doubt, to asthenia of the heart, is not uncommon; indeed it is rather the rule than the exception. But Dr. Kirkes' statement, however, goes much further than this.

I cannot, I repeat, accept the dogma; nay, I strongly and entirely dissent from it, "that absence of murmur is no proof of the absence of even serious organic disease of the valves of the heart."

I maintain that, whilst the *presence* of murmur affords no proof of the existence of structural alteration of the valves, the *absence*, throughout the patient's illness, of murmur or its equivalent prolonged and modified first sound, does afford negative proof in regard to it. In other words, I maintain that whilst murmur may be due to causes other than valvular disease, that the presence of the latter, in a degree capable of deranging health and appreciable to the eye of the anatomist, must, and actually does, always give rise to murmur as long as the heart acts with ordinary vigour.

Hence it will be seen, that I am likewise in disagreement with Doctor Markham, when he says :* "Neither is the absence of murmur any certain indication of absence of valvular disease," etc.

On the whole question here discussed I may remark, that in several examples of chorea given in the sequel, no cardiac murmur, nor other evidence of cardiac disease existed; and that in still greater number, examples are recorded of disease of the mitral valve unaccompanied by chorea.

* *Diseases of the Heart, their Diagnosis, Pathology, and Treatment*, 1856, p. 188.

Doctor Handfield Jones had only one example of valvular cardiac affection in fifteen cases of chorea, and in this exceptional case rheumatism had not preceded. He is of opinion that rheumatic affections rarely tend to choreic complication; but, he admits that chorea and rheumatism are allied, in being both associated with a paretic condition of certain portions of the nervous system; viz., chorea with paresis of the musculo-motor, and rheumatism with paresis of the vaso-motor nerves.[*]

Doctor Hughes, after discussing with great ability the inferences to be drawn from the details of one hundred cases of chorea published by him, in regard to the alleged dependence of that affection upon rheumatic structural lesions of the spinal cord, remarks: " It seems at least doubtful, whether, in most of such cases, there exists anything more than a sympathetic affection of the spinal marrow, as these cases are curable by the same means as others."[†]

Doctor Ogle expresses a similar opinion, adding a number of objections of great weight, as against the doctrine of the rheumatic origin of chorea.[‡]

The frequent occurrence of cardiac murmur in connexion with chorea; the no less frequent association of rheumatism or the rheumatic diathesis, with that disease; and the occasional concurrence of rheumatic arthritis and organic valvular lesions of the heart in choreic subjects, have led many eminent pathologists, as previously shown, to believe that chorea is essentially a rheumatic neurosis, dependent upon eccentric irritation of the heart by rheumatic exudation or deposit upon its valves or external surface; or upon centric irritation of the cerebro-spinal axis itself, by vascular engorgement and thickening of its envelopes, inflammatory softening of its substance, or impairment of its nutrition by capillary embolism.

Is organic lesion of the heart, valvular or pericardial, a usual concomitant; or rheumatism, latent or manifest, a necessary, or even an ordinary precursor of chorea? I confidently answer both these questions in the negative.

* *Clinical Observations on Functional Nervous Disorders*, 1864.
† *Guy's Hospital Reports*, vol. iv., 1846.
‡ *British and Foreign Medico-Chirurgical Review*, vol. xlvi., 1868.

The great majority of my cases of chorea have been in female children under thirteen years, who either have never had rheumatism, or had it at so remote a period, followed by a long interval of general good health, that it could not be regarded as in any degree causative of the chorea, in the absence of organic cardiac disease. In the greater number there was no cardiac mur⁓ mur whatever; and no evidence, of any kind, of organic disease of the heart or pericardium. In a few instances a systolic bellows murmur existed at the apex ; but, unattended with dyspnœa or visceral engorgement, and quickly removed by treatment appropriate to the nervous affection. In nearly every instance, likewise, the attack was traceable to fright or other emotional excitement, or to the presence of intestinal worms. In a few examples the attack was immediately preceded by articular rheumatism, with or without engagement of the heart or pericardium.

Whilst engaged in collecting materials for this work, I did not deem it necessary to record all the cases of chorea in which no evidence of cardiac disease existed. I have, however, preserved notes of a few, which I shall now furnish in abstract. In every example which has come under my notice within the last five years, I have examined the heart, physically, with great care, with a view to determine whether cardiac disease of any kind was present.

Case 1. Mary K., aged twelve years, of sandy complexion and florid face, admitted June 21st, 1869; the daughter of a poor man who, about a year previously, had been an inmate of the hospital, suffering from disease of the aortic valve which has since proved fatal. She had rheumatism the previous Christmas, which confined her to bed, and lasted one month; began to get " fidgetty" three months subsequently. She droops to left side, and admits weakness of left arm and leg ; tongue furred, and suddenly retracted after protrusion ; is very fidgetty even in sleep, frequently starting up. Pulse 108, regular ; heart's action and sounds normal ; *no cardiac murmur*. To have liquor arsenicalis (℔ v. ter die) after meals, and cold shower baths. June 28th ; is much better, less fidgetty. To continue medicine at her home. Owing to scarlatina having appeared in the ward, she was discharged.

Case 2. John M., aged seven, admitted June 13th, 1869. No history of rheumatism ; is very fidgetty and droops to right side. Choreic symptoms well marked, but not in an aggravated form ; *no cardiac murmur*. To have arsenical solution and iron. Discharged cured, June 28.

Case 3. Julia S., aged nine, anæmic, consulted me as an extern patient of the hospital, October 1st, 1869. Her health had been good up to eighteen months previous to the above date; and she never had rheumatism, or passed worms. For the last year and a-half she has been the subject of aggravated chorea. The attacks are in some measure paroxysmal, and during the period of their continuance she cannot speak, and the lips are bitten. Bowels regular; deglutition perfect; pulse 120, regular; action and sounds of heart normal, and *no cardiac murmur*. Muscular power equal on both sides of body. Another child of the same parents, seven years old, has also had chorea, of which she has been cured. To have syrup of iodide of iron and Fowler's solution. Discharged, cured, after a few weeks' treatment.

Case 4. Justin McS., aged nineteen, lithographic printer, admitted February 16th, 1871. Had rheumatism, but without cardiac complication, three months previously, for which he was treated in Baggot-street Hospital. Was noticed to be fidgetty one week after leaving that institution cured; is now sleepless, constantly biting his lips, tossing his arms and legs, and fidgetting with his fingers. Pulse weak, irregular, 96 ; cardiac action irregular and intermittent, but *no cardiac murmur;* sight weak. To have sulphate of quinine and strychnia. Discharged, scarcely improved, after a few weeks' residence in hospital. I subsequently saw this patient in Steevens' Hospital in the same condition as above described.

In rheumatic endocarditis the mitral is the valve engaged in most instances. Yet, in the list of one hundred cases of chorea published by Dr. H. M. Hughes, there were only nine examples of permanent mitral murmur, and, of these, three only were of rheumatic origin. There were, in addition, two examples of temporary mitral murmur, and two of basic murmur, whether temporary or permanent is not stated. Out of fifty-five cases in

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which a cause was assigned for the chorea, fright was the imputed cause in thirty-four instances.

Simple rheumatism was the alleged cause in only five cases; rheumatism with scarlatina in one case, and rheumatism with pericarditis in one.

In Dr. Ogle's Report,* a list of ninety-six cases is furnished, out of which cardiac murmur presumably existed in fourteen instances.† Rheumatism is mentioned as the cause of the chorea in nineteen instances; intestinal worms in fourteen; fright in twenty-three, and menstrual derangement, or sudden arrest of the menses, in eight cases.

Out of a total of one hundred and nineteen cases of chorea published by Hughes and Ogle, in which the origin of the disease has been traced to an assignable cause, fright was the imputed cause in fifty-seven instances, or nearly one-half.

In these cases it cannot be reasonably supposed, in the absence of a rheumatic history, past or present, that rheumatism had any share in the production of the chorea. Rheumatism is mentioned as the cause in only twenty-seven out of the total of one hundred and nineteen cases; and valvular lesion of the heart can have existed in only twenty-three of the whole number, even if we include those cases in which its existence was doubtful.

From the evidence thus summarized, and from my personal observation of chorea, I feel warranted in concluding that it is not a disease of rheumatic origin; neither is it due to valvular lesion of the heart, rheumatic or non-rheumatic. I regard chorea as consisting, essentially, in a state of dynamical instability of the spinal cord, manifested by hyper-impressionability, or excessive irritability of that nerve centre. The sentient and motor tracts within the cranium are likewise engaged, but not the centres of thought or volition. The disease seems limited to the emotional centres, and the conducting media of sensation and voluntary motion.

Anæmia and general debility constitute important factors of

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* *Loco citat.*

† In all the examples where valvular lesion was actually found, I conclude that murmur had been detectable, though not mentioned in the returns. My calculation of totals, which is based upon Dr. Ogle's returns, differs somewhat from his.

chorea, but only as predisponents; and to the state arising from this twofold cause, is due the murmur at the apex, or the base of the heart, when not of direct rheumatic origin.

The basic murmur, when not the result of rheumatic disorganization at the aortic orifice, is purely hæmic, and limited, as such. The murmur so frequently encountered at the apex in chorea, is of strictly dynamic origin, and is caused by atony and partial yielding of the walls of the left ventricle at the acme of systole, as will be presently explained. Rheumatism, by inducing a state of general debility, anæmia, and muscular atony, but to this extent only, may be a remote cause of chorea, and of hæmic or dynamic cardiac murmur, associated or not with chorea; but, inasmuch as the latter association, by dependence on a common cause, is of very frequent occurrence, the mistake is not unfrequently made of assuming the murmur to be organic, and the assumed lesion to which it owes its origin to be the cause of the chorea.

Doctor Walshe* says: "The heart may, probably, also undergo dynamic changes interfering with the closure of its valves, and giving rise to murmurs of the regurgitant class. Thus, a systolic murmur, free from harshness, and of medium pitch, having all the attributes of that denoting mitral regurgitation, is occasionally heard in cases of chorea at the left apex. This murmur disappears as the primary neurotic disease itself disappears; its intensity varies with different beats of the heart; its quality similarly changes; and it may even temporarily become inaudible. The murmur in question is not localized as a hæmic cardiac murmur would be; it cannot be referred to organic change in the mitral valve, seeing that it eventually totally disappears. Nor in many of the cases which I have seen, was there the smallest ground for referring it to latent Bright's disease, or to rheumatic irritation of the endocardium.

"The most plausible hypothesis seems to be that irregular and occasional reflux takes place at the mitral orifice, through disordered action of the muscular apparatus connected with the valve. Just as the external choreal movements sometimes

* *A Practical Treatise on the Diseases of the Heart and Great Vessels,* third edition, 1862, p. 95.

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cease for moments or for minutes at a time, so the cardiac murmurs disappear.

" The main objection to this hypothesis seems to arise from the difficulty of understanding how the heart as a whole can act regularly (as it certainly does in chorea), and yet part of its substance contract irregularly. Still the same contradictory state of things may sometimes be witnessed in the voluntary muscles, portions of which may twitch automatically whilst the remainder of their substance is quiescent.

" The same kind of disorder may conceivably be the cause of certain mitral regurgitant murmurs attending dilated hypertrophy of the left ventricle, and disappearing under treatment. So, too, may be explained a fact, for which it seems otherwise impossible to suggest a plausible theory, that in certain cases of soft, flabby, and possibly fattily atrophous heart, a murmur at the left apex will occasionally be heard, though as a rule completely absent."

Here Dr. Walshe distinctly adopts, though manifestly with misgivings as to its soundness, the doctrine of musculo-papillary spasm, in explanation not only of choreic cardiac murmur, but also of that which is occasionally heard at the left apex in flabby and fatty hearts, and of which no other theory, satisfactory to him, has been offered.

I have already urged a strong, and, as it seems to me, insuperable objection, in terms admitted by Dr. Walshe himself, to the hypothesis of spasm of the papillary muscles in chorea. This objection applies with even increased force to its admission in explanation of dynamic murmur in soft and weakened hearts, in connexion with which no evidence of nervous excitability exists ; and, with great respect for Dr. Walshe, I venture to believe that another and much more rational explanation of these murmurs may be offered, as I will endeavour to show.

Dr. Walshe believes that systolic *basic* murmurs may arise from violent excitement of the heart, either through increased force of the current, or by derangement of its line of transit. This he has witnessed not only in hysterical females, but likewise in nervous male subjects. In hypertrophy with dilatation, systolic murmur may, according to him, be produced at the base, by mis-direction of the current against the edges of the arterial orifices.

I have never met with an indubitable example of dynamic murmur at the base of the heart; all systolic murmurs in this situation, not dependent upon disease of the arterial valves or orifices, are, in my judgment, of hæmic origin ; at least I have never met with any which could not on that assumption be satisfactorily explained.

In 1854 Dr. Stokes,* whilst discussing the subject of functional murmurs, after referring to the opinions of Hope and Walshe who locate them exclusively in the arterial orifices, says: " But we must be cautious in rejecting the opinion that inorganic murmurs may be seated in the mitral orifice. I cannot help believing that I have observed cases of inorganic murmurs, which, so far as physical signs went, were closely similar to those of ordinary mitral disease with regurgitation into the auricles." He observed† prolonged first sound, passing into bellows murmur, in the space midway between the nipple and the left margin of the sternum, in continued and relapsing fever; in measles of asthenic character; and in typhoid variola. He quotes a case of maculated typhus, observed by Drs. Heslop and Lyons in the Meath Hospital, in which, after the first week of convalescence, the first sound of the heart was prolonged ; and in the course of a relapse of brief duration, this prolongation passed into bellows murmur, which continued through a second relapse of ephemeral character, after which the patient, a young female, rapidly convalesced, with complete subsidence of abnormal cardiac signs.

In connexion with this case, Dr. Stokes remarks on the greater frequency of murmur of this character in typhoid than in typhus fever; and proceeds to observe, that " The prolongation of the first sound of the heart, whatever be its cause, is closely related to, though certainly different from, inorganic murmur. It is true that the same general causes seem to give rise to both." He adds: " It appears to arise from irregular contraction of the muscular fibrillæ. In some cases, especially in thin persons, we have found it attended with a peculiar vermicular sensation to the touch, very different from the fremitus of organic valvular

* *Diseases of the Heart and Aorta.*
† *Opus citat.,* p. 428 and 502.

disease. Like the anæmic murmur it subsides during or after the convalescence of the patient, but I think that, when well established, its ultimate disappearance occurs much later than that of a true murmur."

A girl aged twelve, six days ill of simple non-maculated fever, exhibited prolonged first sound; on the ninth day, coincidently with increase of fever, and of the rate and force of the heart's action, this passed into bellows murmur, loudest between the left margin of the sternum and the nipple, but traceable, with progressively decreasing intensity, downwards towards the nipple, and upwards in the course of the aorta; it ceased when the patient sat up. On the eleventh day, with increase of weakness, the first sound and the murmur ceased to be audible, save faintly, behind the sternum. Stimulants were given; the action of the heart improved within an hour; and on the following day both first sound and murmur were again distinctly audible, the pulse being 92. Convalescence now set in; the pulse came down; the murmur gradually faded away; and at the end of a little more than a week, the patient was discharged quite well, the murmur having entirely ceased.*

The preceding observations on the subject of dynamic murmurs possess a very high value, derived from the authority of the writer. They refer to examples of evanescent cardiac murmur, not basic, in association with asthenic fever, gradually disappearing with the progress of convalescence, and vanishing with restored health.

The murmur was, therefore, neither organic nor anæmic in origin.

To what cause, then, is it to be attributed? I hesitate not to answer, to parietal debility of the left ventricle. Of this, but in a minor degree, it furnishes evidence similar to that afforded by suppression of the first sound in low typhus, as noticed by Doctor Stokes.

Doctor M'Dowel has published† several cases of dilatation of the left ventricle, with consequent mitral incompetence and regurgitant murmur, without valvular disease.

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* *Dublin Quarterly Journal of Medical Science*, vol. xiv.
† *Opus citat.*

Doctor Bristowe has likewise published[*] several such cases; but, in some of these there was, in addition to dilatation of the left ventricle, shortening and atrophy of the musculi papillares and chordæ tendineæ, to which, in part, he attributes the mitral regurgitation and systolic apex-murmur. As causes of regurgitation, Bristowe denies the efficiency of anæmia, which "will not explain incompetence," and of fibrinous concretions; and he doubts the occurrence of dilatation of the auriculo-ventricular orifice to a degree sufficient to give rise to reflux. The murmur audible with the first sound and at the apex, in his cases, he attributes to "disproportion between the size of the ventricular cavities and the length of the musculi papillares and chordæ tendineæ."

Doctor Hare detailed before the Pathological Society of London, a case in which he attributed mitral incompetence to lateral displacement of the points of origin of the musculi papillares, and the consequent contraction of the latter in an altered direction.

Doctor Walshe, discussing the subject of mitral regurgitant murmur[†] says: "In rare instances it may be of dynamic mechanism."

Stark [‡] is of opinion that in chlorosis, the fibres of the walls of the ventricles undergo relaxation, in consequence of the temporary derangement of nutrition which defect of red blood cells entails. From this results passive dilatation of the chambers of the heart, and, possibly also, relative inadequacy of the mitral valve.

Doctor Parrot[§] holds that all murmurs designated anæmic, have their seat at the tricuspid, not the aortic orifice; and that a large proportion of murmurs arising in the course of acute rheumatism are produced in the former situation, as the result of debility and yielding of the walls of the right ventricle, of dilatation of the tricuspid orifice, and consequent inadequacy of the tricuspid valve.

[*] *British and Foreign Med. Chirurg. Review*, vol. xx

[†] *Opus citat.*, p 100

[‡] *Gazette Hebdomadaire*, Paris, 1863; and *Archives Générales de Médecine*, 1866.

[§] *Archives Générales de Médecine*, 1866, vol. xi; and *Dublin Quarterly Journal of Medical Science*, November, 1869, p. 590.

Doctor Wilks [*] believes that all mitral systolic murmurs associated with chorea are organic; and the result, not of rheumatism, but of the chorea itself. In all such cases he has found the endocardium in the vicinity of the mitral opening, studded with minute bead-like vegetations of fibrin, disposed in rows.

Da Costa [†] has met with non-organic mitral murmur in cases of obstructed pulmonary circulation; tubercular infiltration; asthma; pneumonia; and in a case of pleuritic effusion, in which, on examination of the body, the valves were found unaltered. He admits the possibility of temporary mitral regurgitation; but considers it not unlikely that many examples of mitral murmur are due to vibration of the blood, produced by increased tension of the mitral valve during temporary excitement of the heart, and favoured by a spanæmic state of the blood.

I cannot admit tension murmurs. I have never met with an example of such. The vibration theory, if correct, should apply to all cases of nervous palpitation in anæmic subjects, and vibration murmurs should be audible in all such cases. Yet how small the per-centage of these cases is, in which mitral murmur is actually present, all clinical observers can testify; and, as Dr. Cuming [‡] justly remarks, the murmur when heard, if due to vibration of the blood, should be conducted by the current into the aorta and be there audible, which is notoriously not the case. The situation of maximum loudness of the murmur indicated by Doctor Da Costa, namely, the third intercostal space, is suggestive of its possible origin in the pulmonary artery; of such a murmur I shall, in the sequel, furnish some examples, and in subjects in whom the absence of organic lesion was legitimately presumable.

Doctor Da Costa is further of opinion, that functional valvular disorders may lead to organic valvular disease; the series being, derangement of the action of the heart, functional inadequacy of the valves, hypertrophy, organic valve-disease.

I fail to perceive how hypertrophy, as such, can lead to

[*] *Medical Times and Gazette*, February, 1869.
[†] *American Journal of the Medical Sciences*, July, 1869.
[‡] *Dublin Quarterly Journal of Medical Science*, November, 1869, p. 591.

organic disease of the valves. In simple hypertrophy, as exemplified in chronic renal disease, no such consequence as disease of the valves is ordinarily met with.

Bamberger is of opinion, that the so-called anæmic murmurs; those which occur occasionally in fever, and many of those which are developed in the course of rheumatism, are veritable mitral reflux murmurs; and that they are due to a weakened state of the papillary muscles, in consequence of which the valve is not rendered sufficiently tense, and after vibrations are permitted.

M. Raynaud* contrasts non-organic and organic murmurs as follows: viz., "In the first (non-organic) the seat is at the base of the heart; it is propagated in the direction of the ascending aorta; it is soft and intermittent. The second (organic) is rougher; it assumes to some extent the character of the *bruit de scie, bruit de lime,* or *bruit de râpe;* it is more fixed, and, if it occupy the aortic orifice, the lesion is well pronounced, and possesses other characters."

It can now, however, be no longer maintained that the seat of non-organic murmur is exclusively basic. Nor, indeed, that murmurs of organic origin are invariably rough, and exhibit the characters above mentioned.

Doctor Peacock has given a list of cases, five in number, in which mitral regurgitation was announced by systolic murmur at the apex; but, in four of these cases, there was, in addition to dilatation of the mitral orifice, disease of the mitral valve in greater or less degree. In the fifth case there was adhesion of the pericardium, with left ventricular hypertrophy and dilatation, and enlargement of the mitral orifice, but "no material valvular disease."†

That non-organic mitral regurgitant murmur may arise from causes other than those above mentioned, I have, in another place,‡ endeavoured to show. I have given cases to illustrate the occurrence of such murmur in connexion with anæmia, purpura, masturbation, and excessive tobacco-smoking. To this

* *Nouveau Dictionnaire de Médecine,* p. 388.
† *On Some of the Causes and Effects of Valvular Disease of the Heart,* 1865.
‡ *British Medical Journal,* November 16th and 23rd, 1867.

list I would now add chorea, and weak and fatty heart (see cases of J. O'B. and R. Burrows). It is quite true that in none of these cases, except those referred to under the last-mentioned two heads, has the negative diagnosis in relation to organic disease been confirmed by the evidence of *post mortem* examination of the body; because no opportunity for such was presented. But the cessation of the murmur under treatment, coincidently with the disappearance of irregularity in the heart's action, and the general improvement of the patient's health, constitute presumptive evidence amply sufficient to warrant the diagnosis given.

In the paper above referred to, I have expressed the opinion, " that an explanation of the occurrence of murmur under the circumstances now stated, must be sought mainly in physiological considerations having reference to the mode of combination, the direction, and the relative force of contraction of the *musculi papillares* of the left ventricle. That a yielding of a particular portion of the walls of the ventricle during the centripetal movement which takes place in the act of contraction, may so alter the direction in which one or both of the *musculi papillares* act upon the segments of the mitral valve, as totally to invert their function, by rendering them effective agents, not in closing, but, in *opening* the orifice of communication with the auricle, seems legitimately presumable from what occurs in the right ventricle, under similar circumstances, as shown by the late Mr. W. King.

" Such yielding may be due, not as in the right ventricle, to simple engorgement, but either to a want of sufficient contractile power in a particular part of the walls of the ventricle, or to ataxy or want of co-ordination in the contraction of the different portions of those walls.

" Without committing myself to a definite theory on this subject, I may state shortly the view which I entertain as to its pathology. The musculi papillares of the left ventricle (as also of the right) are so placed that in the state of complete ventricular systole they interlock, or pass to opposite sides of the chamber. In virtue of this interchange of position they so act upon the segments of the mitral valve, that the lines or

axes of their contraction decussate at an acute angle, and the valves are drawn, each towards the opposite side of the ventricle. Such a movement can take place manifestly only at the acme of ventricular systole, causing *active* closure of the valves, tension of these and of the chordæ tendineæ, and the sharp " click " constituting the terminal portion of the first sound. If from any cause whatever, involving inability of the ventricle to empty itself, such as yielding of a particular portion of its walls during their centripetal movement in the act of systole, the musculi papillares are prevented from performing the movement of transposition already indicated, they will act upon the valves, *not* in the direction of the opposite wall of the ventricle, but towards that from which they take their origin, and will therefore divaricate the valves, and permit regurgitation.

" It has been objected that as, according to this view, the closure of the valves is effected only at the acme of systole, regurgitation may have taken place previously to its occurrence.

"But it should be remembered that the closure of the valves is passive, *quoad* the pressure of the mass of blood in the ventricle at the commencement of systole, and that their active closure is terminal, and rather an act of tension to prevent their being reversed at the acme of ventricular systole. If, at this moment, when the chordæ tendineæ are made tense by the contraction of the corresponding papillary muscles, one of them is made to act in the direction of the attached margin of the valve with which it is connected, owing to an irregular or centrifugal movement of a portion of the wall of the ventricle, the segment of the valve so acted upon is of necessity raised off the auriculo-ventricular opening, and regurgitation takes place at the close of systole.

"It is worthy of remark, as bearing upon this view, that regurgitant murmurs of this character are always, so far as I have observed, postsystolic, or occupy the terminal portion of the period of systole."

Finally I draw the following conclusions:

" 1. That regurgitation may take place at the mitral orifice independently of disease of the valves, and of dilatation of the orifice of the ventricle.

" 2. The murmur which announces this form of regurgitation is faint and abbreviated, usually confined to the area of the apex, and not associated with embarrassment of respiration, vascular turgescence, or the other usual signs of organic mitral regurgitation.

" 3. The pulse associated with this murmur is quick, abrupt, and feeble; and either rhythmically irregular, or of the alternately ascending and descending character previously described. .

" 4. Examples of this murmur may be met with in those who are the subjects of anæmia, or purpura; or who have become enfeebled by masturbation, or by the excessive use of tobacco."

To the list above given I would now add chorea, and fatty softening of the left ventricle, as previously stated.

With the experience I have since had, I incline to endorse the opinions thus expressed seven years ago, and am satisfied, whatever may be the value of the theory propounded, that causes, of which the above are examples, which debilitate the ventricular walls, may act indirectly as causes of mitral reflux of uncertain duration, but generally amenable to treatment.

Doctor Cuming of Belfast, has published,* in a paper of great practical value, the particulars of two cases of similar import to the foregoing. In both, systolic murmur confined to the mitral area existed; and in one of these (case of R. C.) pulmonary and general vascular engorgement with anasarca were present; the patient died; there was no dilatation of the left ventricle, no atrophy of the musculi papillares or chordæ tendineæ, and no disease of the mitral valve, or only a slight warty growth upon it quite inadequate to cause regurgitation. The muscular structure of the heart is reported as healthy; but, microscopic examination of it, which constitutes the only reliable evidence, negative or positive, on this subject, is not mentioned. This case is the first, as far as I am aware, in which mitral reflux murmur, accompanied by the usual symptoms of advanced organic disease; viz., pulmonary engorgement, hæmoptysis, and anasarca, existed; and in which dissection demonstrated the

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* *Dublin Quarterly Journal of Medical Science,* May, 1868.

absence of anomaly or disease in the cavities, walls, or valves of the heart. I have, since that date, met with two cases similar in their principal features to the foregoing, which, with the results of *post mortem* examination of the bodies, are given in the sequel (cases of J. O'B. and R. Burrows).

In Dr. Cuming's second case the patient had been weakened by purging; no antecedent murmur existed, as he had been examined and passed for a life assurance office. At the period of report, a soft bellows murmur of systolic rhythm and mitral diffusion existed; but it ceased after brief tonic treatment, coincidently with restoration of perfect health.

This case presents many features of resemblance to those published by me in the preceding year, and to which Dr. Cuming refers. He, however, misapprehends the meaning I desired to convey when I stated that the absence of pulmonary congestion in my cases was, *pro tanto*, evidence of the absence of valvular disease. I did not, by any means, intend thereby to intimate that pulmonary and systemic engorgement, followed by the usual results, may not occur in such cases. I only wished to point out by the observation alluded to, the temporary character, and, inferentially, the non-organic cause of the murmur in my cases.

I am entirely of Dr. Cuming's opinion when he states that " it is very probable that several of Dr. Hayden's cases would have exhibited signs of pulmonary and systemic congestion had the regurgitation not yielded to treatment."

From what has preceded, I feel warranted in concluding that a a murmur, postsystolic in time; in continuity with the first sound; extending to a variable length into the short pause; loudest at the apex, to which in most cases it is confined; loudest, likewise, in the recumbent posture, and diminished in intensity, or temporarily suspended, when the erect posture is assumed; unaccompanied by the symptoms and signs of pulmonary congestion, and by outward displacement of the apex-beat; and associated with rapid changes in the rate of cardiac and radial pulsation, irrespectively of physical exertion, indicates mitral reflux *without* structural lesion at the mitral orifice, or permanent dilatation of the left ventricle. I further hold, but not with equal confidence,

that in such a case the walls of the left ventricle would be found in a softened or atonic condition, and that to this, as previously explained, the murmur would be due.

The murmur is faint, soft, and blowing in character; and, in the instances which have come under my notice, has fallen short of the second sound; although it is conceivable that such should not invariably be the case.   In the two examples above referred to, in which I had the advantage of a *post mortem* examination of the bodies, the result as regards the state of the valves was negative.

Doctor Nixon recently read before the Medical Society of the College of Physicians a paper on this subject, which was characterized by very considerable research and ability.*   He submitted the details of five cases, in three of which he had the advantage of examining the body after death.   In all three the mitral valve was unaltered, and apparently adequate to close the orifice ; but, in two of these, the muscular structure of the left ventricle was in a state of fatty degeneration.   The third was an example of chronic meningitis and general mal-nutrition.   Of the two cases in which death did not follow, one was that of a delicate female, who had been greatly reduced by flooding, during a miscarriage, eight months previously, and in whom both a basic and an apex-murmur existed, which ceased under treatment.   The other was that of a young lady, who exhibited a murmur, only during nervous palpitation following mental disturbance or the use of green tea.

It will be observed, that in all these cases, except the last, there was general debility ; and, in two of the number, the left ventricle was proved, by dissection, to be in a state of fatty softening.   In the case of the young lady, nervous debility may be inferred.

Doctor Nixon is of opinion, that the conditions under which functional mitral murmur has been met with are so various, that no single explanation of the mode of its occurrence will suffice ; but that, in most cases, it is caused by derangement of the consentaneous action which normally subsists between the muscular walls of the left ventricle and the papillary muscles of that

* *Dublin Journal of Medical Science,* June, 1873.

chamber; and that this is due "to some defect in the vital power or condition of the heart itself, which leads either to atony of the papillary muscles, or derangement in the rhythm of their movements." I believe that in most cases, if not in all, debility of the walls of the left ventricle may be reasonably supposed to exist, and that the hypothesis which I have ventured to propound affords not only the most generally applicable, but the most rational explanation of the phenomenon.

Doctor Nixon's observations as to the differential diagnosis of this murmur are most judicious. The features upon which he relies, as of positive diagnostic value, may be briefly set forth as follows:

1. The murmur is intermittent, and variable as to intensity.
2. Usually present in the recumbent posture, it generally ceases when the patient sits up.
3. It is loudest, not at the apex, but over the body of the ventricle (Da Costa).
4. There is usually absence of pulmonary distress, and of accentuation and doubling of the second sound.
5. There is no alteration in the size or position of the heart.
6. The radial pulse is variable as to rate and volume.

The foregoing observations are of the utmost importance. I am not in a position to affirm all the rules just laid down for testing this murmur; but I have no hesitation in subscribing the majority of them, and in declaring that Dr. Nixon's paper is, in my judgment, the most valuable contribution hitherto made towards the differential diagnosis of functional murmur at the mitral orifice.

Skoda, writing of the diagnostic value of accentuated second sound in the pulmonary artery, as positive evidence of regurgitation at the mitral orifice in cases of systolic murmur at the apex, says:* "If the force of this sound be not increased, the murmur then indicates that the surfaces of the valves, or the lining membrane of the ventricle about the arterial openings, are roughened."

Doctor Todd, likewise, held the opinion that a rough condition of the ventricular surface of the mitral valve was competent, of

* *Opus citat.*, p. 229.

itself, to give rise to systolic murmur in the mitral area, liable to be confounded with that of mitral reflux.*

He says : " But if the deposit take place on the *ventricular* surface of the valve (and it generally does so on the ventricular surface of its inner curtain) then you have no disturbance of valvular function. In both cases, however, you have a systolic bellows-sound, and in both cases that sound is best heard at the apex of the heart."

In the pseudo-murmur, however, the absence of Skoda's sign of accentuated second sound; the murmur being audible towards the base ; and inaudible, or very faintly audible beneath the left scapula, suffice, in his opinion, to establish the distinction between the two cases.

Doctor Walshe also believes† that " vegetations on the ventricular surface of the (mitral) valve " are competent, without causing inadequacy or mal-adaptation of the valve, to produce systolic murmur at the apex.

The concurrence of so many eminent authorities in support or acceptance of the doctrine of systolic murmur of maximum intensity at the apex, due to simple roughening of the ventricular surface of the mitral valve, or of the adjacent portion of the ventricular wall, and liable to be confounded with that of mitral incompetency, would seem to constitute it an established dogma in cardiac diagnostics.

Nevertheless, I venture to question its soundness, and to express a doubt that it rests upon stronger evidence than *a priori* reasoning. At least, I have never fallen upon the details of a case, in which murmur of the site and rhythm above mentioned, viz., " systolic murmur best heard at the apex of the heart," was satisfactorily traceable to the cause now alleged, to the exclusion of all others competent to produce it. Nor have I met with a single example, in the living, which would warrant me in assigning to the alleged murmur, even provisionally, a place in cardiac pathology. I have, however, met with an example of *basic* systolic murmur due to roughening of the ventricular surface of the mitral valve.

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* *Clinical Lectures,* second edition, 1861, p. 77.
† *Opus citat.,* p. 99.

Doctor Todd lays too much stress, in regard to differential diagnosis, upon the fact that this murmur is heard but feebly, or not at all, at the left scapula; on the absence of intensified second sound; and on the less degree of disturbance of the heart and general distress which accompany it, as compared with the murmur of veritable mitral regurgitation.

I have repeatedly found indubitable mitral regurgitant murmur of organic origin, totally inaudible at the left scapula and unaccompanied by intensified second sound. Indeed, such is the rule in the case of soft murmurs of recent origin, and occurring in a feeble heart before appreciable derangement of the pulmonary circulation has supervened. As above shown, these negative signs and symptoms are characteristic of functional mitral murmur.

Dr. Todd indicates, however, the true distinction between systolic murmur at the apex due to simple roughening of the ventricular surface of the mitral valve, and the murmur of mitral reflux to whatever cause due, when he says that the former "may be heard well up to the base of the heart." Not only may it be so heard, but it is absolutely loudest at the right base, and audible throughout the arch of the aorta and its primary branches with great distinctness If, with such a murmur, likewise, but less distinctly, audible at the apex, and unaccompanied by the other evidence of mitral regurgitation mentioned by Dr. Todd, the second sound in the aorta be unimpaired and free from murmur, the diagnosis of a systolic murmur of efflux in the aorta from roughening of the ventricular surface of the anterior mitral segment, or of the adjacent portion of the ventricle, may be positively made.

Pseudo-murmurs are sounds simulating cardiac murmurs, or liable to be mistaken for them. They arise from two sources; namely, pericarditis in the "dry" stage, and exocardial pleuritis.

The attrition-murmur of pericarditis, before it has assumed a character of decided harshness, may closely simulate veritable cardiac murmur. That is to say, in the early stages of the affection, when the free surfaces of the serous membrane, either by congestion of its vessels and arrest of its normal secretion, or

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by fibrinous exudation as yet soft and plastic, cease to glide noiselessly over one another, as they do in a state of health, during the alternate movements of the heart in contraction and dilatation.   The resemblance will be the stronger, and the diagnosis proportionately more difficult, when the attrition-sound is localized at the apex of the heart, as it occasionally is in the first instance.   I have had, on two or three occasions, the opportunity afforded me of studying a pericardial friction-sound, limited to the apex, in its nascent stage (see case of John Clinton).   Such murmurs are usually soft, and of single and systolic rhythm, during at least the first twenty-four hours of their existence, and at this stage are not readily distinguishable from regurgitant mitral murmurs.   They are, however, more superficial in site, as indicated by their proximity to the ear of the observer, and less constant in character, undergoing a change in rhythm and in quality within the first two days.

The ordinary friction-sound of pericarditis is, however, of double rhythm, not strictly coincident in time with the sounds of the heart as alleged by all writers on this subject, with the exception of Dr. Walshe and Dr. Hyde Salter.*   The first attrition-sound coincides with the impulse and the first sound of the heart, and is therefore in the strictest sense systolic.   The second, however, coincides, not with the second cardiac sound, but with the early portion of the succeeding or long pause, and is strictly postdiastolic in rhythm.   I have met with not more than two exceptions to this rule, which may, therefore, be regarded as constituting an important element of distinction between the frottement of pericarditis and double aortic murmur. In the exceptional instances referred to, the second friction-sound was strictly diastolic, and, therefore, not by rhythm distinguishable from the bruit of aortic reflux.

Doctor Walshe says : " When of regular rhythm, the friction-murmur falls a little after the corresponding valvular sound. But such regularity as this is the exception, not the rule ; the friction-murmur may be very distinct during the postdiastolic silence, and occasionally falls between the first and second heart sounds.   In point of fact, want of distinct synchronism of the

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* *Opus citat.*, p. 112, and *Lancet*, July 29th, 1871.

murmur with either of the two sounds is a very habitual and distinctive attribute."*

Sir T. Watson describes the acoustic phenomenon of pericarditis as a "to-and-fro" sound, as "of two surfaces rubbing backwards and forwards against one another."† This is a just comparison, and a most accurate description of the sound.

Collin‡ compared it to the creaking of new leather, and named it, from the resemblance, *bruit de cuir neuf.* He says: " Perhaps this sound may be a constant symptom of pericarditis before the existence of effusion into the serous envelope of the heart, a symptom very fugacious in cases where this disease has terminated in a few days, of longer duration when it is chronic." He adds : " I explain, then, this phenomenon by the rubbing of two layers of dried serous membrane. This kind of desiccation appears to be the first effect of inflammation upon membranous tissues, etc."

Broussais says:§ "There is a phenomenon worthy of attention, to which, perhaps, it has not been given in a sufficient degree; this is the noise of parchment, which may be distinctly perceived by means of the stethoscope. Whilst exploring with this instrument in incipient pericarditis, one experiences a sensation such as that which might be yielded by two dry bodies, for example, of parchment, whilst rubbed one against the other; and this sign, when combined with the pain and anguish, should leave no doubt as to the existence of inflammation."

It would thus seem that Broussais fully appreciated the diagnostic value of friction-sound as a sign of pericarditis.

Stokes, however, was the first writer who placed in a clear light the pathological significance of pericardial friction-sound.‖ By a course of rigid observation of the vital phenomena of the disease in a number of typical and highly illustrative examples, followed by careful dissection of the bodies, he conclusively

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* *Opus citat.,* p. 112.

† *London Medical Gazette,* vol. xvi., part i., April 11th, 1835.

‡ *Les Diverses Méthodes d'Exploration de la Poitrine, etc.,* par V. Collin, Paris, 1824.

§ *Commentaires des Propositions de Pathologie,* 1829, tom. i., p. 398.

‖ *Dublin Journal of Medicine and Chemical Science,* vol. iv., 1834; published September 1st, 1833.

established, not only the diagnostic value of friction-sound as a sign of pericarditis, but distinguished it by salient characteristics from other acoustic phenomena in the region of the precordium, of different origin and significance, with which it is liable to be confounded.   As distinctive signs of this sound, he dwells upon the modification which it undergoes in consequence of the local or general abstraction of blood; its sudden supervention, variable character, and rapid change of situation; its occurrence with *both* sounds of the heart, where no sign of organic disease had previously existed; its subsidence under treatment, and not again becoming audible under excitement.   He dwells likewise upon tactile vibration or *frémissement*, as distinguishing pericarditis from valvular lesion.

I cannot attach equal value to this last mentioned sign as distinctive of pericarditis.   I have repeatedly observed *frémissement* of great intensity associated with valvular murmur exclusively, most frequently with that of presystolic rhythm, indicating obstruction at the mitral opening.   No doubt, the fremitus of pericarditis is usually double, corresponding in that respect to the friction-sound; but pericardial murmur is occasionally single, and, when so, it is usually systolic, and located at the apex.   In such a contingency fremitus might indicate, and numerically in about equal proportion, either constriction of the mitral orifice, or pericarditis at the apex.   In a small per-centage of cases it arises from mitral regurgitation.   The mode of distinguishing these phenomena from one another will be pointed out further on.

Doctor Stokes very appropriately designates as "latent pericarditis" that form of the affection, of which the only evidence afforded consists in friction-sound, with or without *frémissement*.

Pericardial friction-sound is notably variable in character, in site, and in rhythm.   It is likewise fleeting, or liable to temporary suppression.

The *quality* of the sound is in most instances harsh and unpleasing, and its *pitch* may vary, even in the same case, between the limits expressed by " grating " and " filing."

When these variations are presented in the same person, they

are noticeable from day to day, the changes being seldom more abrupt, and they are usually attained, by gradations, when the extremes above mentioned are reached, which, however, is not often the case.

The *site* of pericardial murmur is usually basic; but it may extend over the entire precordium, or it may be limited to the apex; and the relative frequency of the three sites would be indicated by the order in which they are above enumerated

The existence of this murmur by preference at the base, is explained by the closer proximity of the visceral to the parietal pericardium in that situation, owing to the peculiar configuration of the heart. To this circumstance is likewise due the fact that, at the base, friction-sound is always of double rhythm. The transfer of the phenomenon from base to apex, or *vice versa*, is only apparent. Usually, the cases which present it most prominently, are those in which friction had been previously audible over the entire precordium; and in these the apparent transfer is due to suppression of the sound in one of the two situations mentioned. Occasionally, however, such is not the case, and a veritable extension of inflammation, to a previously unaffected portion of the pericardium, coincides with adhesion in the previous situation of friction-sound.

The rhythm of pericardial friction-sound depends upon the movements, not the sounds, of the heart. It is, therefore, as already stated, usually double at the base; because, in this situation, there are two periods in the cycle of cardiac movement when the visceral and parietal layers of the pericardium are in mutual contact; one, corresponding to ventricular systole, during which there is an upward movement with tension of the ventricles, and repletion with tension of the auricles, and the other coinciding with a downward movement or subsidence of the ventricles in diastole, and a simultaneous vermicular or vibratory contraction of the auricles. The first element of the double sound is therefore strictly systolic, sharp, and abrupt, because of the single and energetic character of the ventricular movement upon which it depends; whilst the second is postdiastolic, or posterior in time to the second sound of the heart; less loud, and of a prolonged and shuffling character, in accordance with

the weaker, more protracted, and somewhat undulatory movement to which it owes its origin.

Thus, a double pericardial friction-rhythm is recognised; namely, ventricular systolic, and ventricular diastolic; with the latter auricular systole coincides. The character of ventricular systolic frottement is uniform; not so, however, that of ventricular diastolic rhythm, because of its compound origin. The latter is usually composed of a succession of short and abrupt "rubs," commencing, at the base, *after* the second sound, but occasionally coinciding with it; and sometimes, though rarely, exhibiting by one of its elements, a distinct presystolic rhythm.

In my lectures on "Mitral Obstruction,"* I urged the possibility of mistaking pericardial friction-sound, possessing this rhythm exclusively, for true endocardial presystolic murmur. But, inasmuch as such rhythm, even in combination, is rare in pericarditis, and still more so in the isolated form, the error of diagnosis adverted to is not likely to be committed.

In a very able lecture, published by the late Dr. Hyde Salter,† he described a pericardial friction-sound of triple rhythm; namely, ventricular diastolic, auricular systolic, and ventricular systolic. The second mentioned he truly alleges to be presystolic in time, and liable to be confounded with the endocardial murmur of mitral constriction. He gives, in illustration, the particulars of four cases, which may be summarized as follows:

Case 1. Triple frottement, which by-and-by became double by loss of one element (which, in particular, is not stated); auricular systolic friction-sound of presystolic rhythm existed at the inner part of the right third intercostal space; and, after death, this was found to depend upon a rough deposit of fibrin upon the right auricle. In the progress of this case, both elements of the double friction-sound at the base ceased simultaneously.

This is certainly not in agreement with my experience, which is to this effect, that double friction-sound at the base, in process of abolition, becomes single, for a brief period, by loss of one of its elements previously to final cessation. I cannot recall a single exception to this rule.

Case 2. Female; albuminuria; presystolic murmur over left

* *Medical Press and Circular*, July, 1866.   † *Lancet*, July 29th, 1871,

third costal cartilage, one inch from the sternum, soft, single, and audible only within an area not larger than a halfcrown piece at the point indicated. On dissection, the left auricle only was found covered with rough lymph, and this coincided exactly with the seat of friction.

Case 3 A female; albuminuria and bronchitis, with cavity in right lung; frottement at inner part of right third intercostal space, loudest within an area of the size of a shilling, and audible over a space not larger than a crown piece. It was double; the first part rough and presystolic, the second fainter and softer, and systolic in rhythm. He concludes that the first was auricular systolic, and the second auricular diastolic. After a few days, friction became general over the heart. Autopsy: Recent general pericarditis, with effusion of soft lymph.

Case 4. A lady; sub-acute rheumatism; double frottement in right third intercostal space near the sternum, and limited in extent. The first was the louder of the two, and presystolic in rhythm.

Doctor Hyde Salter has demonstrated the truth of a theorem which I have long held to be demonstrable, but have never had the opportunity of proving; namely, that the rhythm of pericardial friction-sound is as the natural movement of the portion of the heart engaged, and the mobility of the opposed surfaces; and hence, with a knowledge of the movements of the different portions of the heart in health, a diagnosis of the seat and limitation of frottement may be made; and, conversely, that by accurate observation of the seat and rhythm of friction, the natural movements of the heart may be determined.

When the phenomenon is about to be suspended in consequence of liquid effusion into the pericardium, or to be suppressed by adhesion of its opposed surfaces, the friction-sound, even at the base, becomes irregular and, still later, single in rhythm (Case 13, Jane Armstrong).

At the apex a double friction-sound is rarely produced; and of this an explanation may probably be found in the peculiar character of the movements of this portion of the organ. In systole the apex moves upwards and forwards abruptly, and with much energy, gliding forcibly along the

anterior wall of the pericardium ; but, in diastole it recedes from the anterior wall, sinking, by its own weight, to the fundus of the pericardium ; hence, pericarditis at the apex is usually announced by a single and systolic friction-sound The exceptions are of two kinds; namely, where the posterior surface of the apex is also coated with lymph, and therefore competent to educe friction-sound in its descent. Lymph, however, is very rarely effused upon the posterior surface of the apex, and hence frottement of diastolic rhythm at the apex is of exceptional occurrence. Double *bruit de frottement*, of basic origin and great intensity, may be likewise audible at the apex by transmission ; but, its relative loudness at the apex and the base, respectively, or its point of maximum intensity, would, in such case, be a sufficient guide to the seat of its production.

Pericardial friction-sounds exhibit, in the majority of instances, what Dr. Stokes very appropriately designates as a " respiratory rhythm ;" that is, a rhythmical intensification corresponding to the acts of inspiration. This, he thinks, may be due either to descent of the diaphragm in inspiration, or to pleuritic friction at the lower and anterior portion of the left pleura. I cannot, however, admit either of these explanations as satisfactory. The cordiform tendon of the diaphragm, to which the pericardium is attached, is all but motionless in respiration, as shown by the unaltered position of the apex during the fullest inspiratory effort ; and the seat of greatest intensity of the inspiratory frottement is not the lower, but the upper portion of the precordium. The rhythmical intensification of pericardial friction-sound, corresponding to inspiration, is due, I doubt not, to the advance of the anterior edge of the lungs between the chest wall and the pericardium during inspiration, by which the opposed surfaces of that membrane are brought into more forcible contact. If, from any cause whatever, as, for example, pleuritic adhesion, this advance of the lungs should not take place, I venture to assert that in such a case the pericardial friction-sound would be devoid of inspiratory rhythm.

The area of diffusion of these sounds is usually circumscribed, or nearly so, by the limits of the precordium; a characteristic by

which it is distinguished from all other acoustic phenomena
of cardiac origin. In children it is usually audible over the
entire front of the chest; and also, but less frequently, to a
corresponding extent in the back, owing to the high conducting
properties of the thorax at this period of life. It attains its
maximum of intensity, and is most extensively diffused, when
pericarditis supervenes upon ventricular hypertrophy (see case
of Clinton).

In females, owing to the same cause as in children, Dr. Stokes
surmises friction-sound may be likewise more extensively audi-
ble. I cannot say that I have been able to confirm this obser-
vation. In advanced age, and in gouty subjects, the chest walls,
rendered more resonant by ossification of the costal cartilages,
and by partial ankylosis of its articulations, may likewise serve
as better conducting media for sound; especially that having
the character of frottement, which involves perceptible vibra-
tion.

The *cause* of pericardial friction-sound is regarded by the great
majority of writers on this subject, as consisting in plastic fibri-
nous deposit upon one or both surfaces of the pericardium, gene-
rally both. Collin, however, held that it was due, exclusively,
to the mutual friction of the opposed pericardial surfaces in a
state of dryness from arrest of the normal serous secretion. His
explanation of the phenomenon is given at page 291.

Hope advocated a similar view, but less exclusively and dog-
matically. He was of opinion " that deficient lubricity of the
pericardium from defective secretion in the earliest stage of in-
flammation, may possibly be *one* of the causes of the creaking
sound, independent of lymph."[*]

Cessation of pericardial frottement may be due to cohesion of
the opposed serous surfaces; or, to serous, sero-sanguineous, or
purulent effusion separating them. Under the influence of these
several causes, the suspension or suppression of the pseudo-mur-
mur is gradual, and in the process its rhythm and force are much
modified. If adhesion take place, the friction-sound is perma-
nently suppressed in the seat and to the extent of adhesion; and

* A Treatise on the Diseases of the Heart and Great Vessels, third edition, 1839,
p. 168.

its cessation above, whilst it continues to be audible at the lower portion of the precordium, is conclusive evidence of progressive adhesion, commencing superiorly. An inversion of this order, however, would not be of similar import, because the cessation of friction-sound progressively from below upwards might be due, either to adhesion or liquid effusion; and the differential diagnosis would rest upon other evidence, mainly that yielded by percussion.

The friction-sound of pericarditis is liable to be confounded with:

1. Double aortic murmur.
2. Single (systolic) aortic murmur.
3. Systolic mitral murmur.
4. Left pleuritic frottement with short and rapid breathing.
5. Left pericardial pleuritic frottement, synchronous with the impulse of the heart.

The features of resemblance between double valvular murmur at the aortic orifice, and to-and-fro pericardial friction-sound at the base, are, the reduplication of murmur in both alike, the alleged synchronism of the murmurs with the normal sounds of the heart, and the maximum intensity of both at the base.

The aortic murmurs, however, are transmitted upwards in the course of the aorta, and, of these, the systolic likewise into the carotid and subclavian arteries; they fade *gradually* away, and equally in all directions except in the line of the aorta, as the stethoscope is shifted from the point of their greatest intensity. They are strictly synchronous with the sounds of the heart in the great majority of cases; they do not vary in rhythm, and, though loud, they are manifestly not of superficial origin; they are not modified by ordinary treatment, nor are they intensified by pressure made upon the precordium.

Pericardial friction-sounds, on the contrary, abruptly terminate at the limits of the precordium, except in children and in cases of great enlargement of the heart; they are not audible to a greater distance in one direction than another; they are never transmitted into the neck; they are not both synchronous with the sounds of the heart, the second being, as already stated, post-diastolic in time; they are peculiarly variable as to rhythm,

quality, intensity, and situation; superficial as to origin; much intensified by surface pressure; and readily modified by treatment. Finally, they most frequently exhibit an inspiratory rhythm, or periodic intensification corresponding to the inspiratory act; * and are usually associated with *frémissement*. This latter is a most valuable indication, though not pathognomonic of pericardial friction. It is much more frequently met with in association with the latter than with valvular lesion; indeed, the friction-sound of pericarditis is never unaccompanied by fremitus, save in its nascent stage, and in that immediately preceding its final extinction, when it is soft and faint.

Frémissement cataire is one of the most remarkable phenomena exhibited by the heart in a state of disease. It has been aptly compared by Laennec, to the sensation communicated to the open hand placed upon a cat's back whilst the animal purs; and it usually coincides in rhythm and varies in intensity with the murmurs audible beneath, the harsher murmurs always yielding the strongest tactile vibration. It may be most readily perceived by means of that portion of the palm corresponding to the roots of the fingers, placed with moderate pressure over the seat of friction.

I have found *frémissement* associated, not only with pericardial attrition-sound, but likewise with valvular murmur of every kind, except those of single and diastolic rhythm at the orifice of the aorta, or the pulmonary artery. In both these cases I conclude that arterial systole, though capable of producing a murmur of reflux, is incompetent to develop a force sufficient to educe thoracic vibration.

I have already intimated that, exceptionally, the friction-sound of pericarditis may be diffused extensively, and even universally, over the chest, in children, and likewise in adults who are the subjects of cardiac hypertrophy. In the latter cases I have heard

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* This sign, which is due to the pressure of the pericardium against the anterior surface of the heart by the advancing edges of the inflated lungs, is, when present, pathognomonic of pericarditis; but it fails where vesicular emphysema of the edges, or great enlargement of the heart exists; because, in the former case, the edges of the lungs are permanently inflated and overlie the pericardium; and in the latter they are permanently displaced outwards, and cannot advance during inspiration from want of space. Permanent adhesion of the lungs to the sides of the pericardium will likewise neutralize this sign.

it beneath both clavicles, on both sides of the back, and along the dorsal spine. In children loud valvular murmurs are no less extensively diffused. Nevertheless, attention to the preceding distinctions, as well as to those of a general kind, to be hereafter mentioned, having reference to the pulse, the seat of apex-pulsation, and the area of precordial dulness, by which double valvular disease at the aortic orifice is characterized, will suffice to establish the differential diagnosis between murmurs due to that disease, and the pseudo-murmurs resulting from pericarditis.

Aortic murmur of single and systolic rhythm is liable to be confounded with bruit de frottement, only when the latter approaches extinction by cohesion of the opposed surfaces. At this period the friction-sound of pericarditis is usually, even at the base, single and systolic in rhythm, less loud and harsh than previously, and, therefore, more closely similar in quality to the murmur of valvular obstruction. The latter, however, is *always* propagated in the line of the ascending aorta, and transmitted into the carotids; the former *never*.

From the murmur of mitral reflux, pericardial friction-sound of single and systolic rhythm and apex origin, may be distinguished, chiefly, by the positive signs which characterize the former; namely, its being transmitted into the axilla, audible, in most cases, at the inferior angle of the left scapula, and associated, after the initial period, with intensification of the second cardiac sound in the pulmonary artery, together with the signs of pulmonary congestion, and engorgement of the right side of the heart.

Indeed, by no other positive characteristics than its unsteadiness as to quality, site, and rhythm, and its brief duration, can single systolic friction-sound at the apex be distinguished from mitral reflux murmur; but these, in conjunction with the above given character of mitral reflux murmur, will justify a confident diagnosis, even in the most obscure cases, after careful examination once or oftener repeated.

From the respiratory frottement of pleuritis of the left side, the friction-sound of pericarditis may be readily distinguished, by causing the patient to suspend respiration for a moment. The procedure will, of course, likewise suspend the friction-

sound, if it be of pleuritic origin ; not so, however, if its seat be the pericardium.

The distinction between the attrition-sound of pericarditis, and that of pleuritis engaging the pericardial portion of the pleura and adjacent edge of the lung, and deriving its rhythm from the movements of the heart, not from those of the lung, is attended with the utmost difficulty.

Doctor Stokes* states that in the latter case, the murmur is loudest rather outside than within the limits of the precordium. I can confirm the accuracy of this distinction.

Doctor Mayne,† discussing the positive value of frottement synchronous with the action of the heart, as a sign of pericarditis, inquires : "But even in simple pleuritis may not the mechanical impulse of the heart against the lung, when covered by inflamed pleura, produce a distinct frottement synchronous with the systole of that organ, and thus cause difficulty in the diagnosis, unless general symptoms are taken into the account ?" To this I answer, that in such a case friction-sound would be confined to the apex, and either inaudible at the right base and in the middle line, or audible in these situations only by transmission.

In the course of pericarditis, a systolic bellows-murmur may be developed at the base or at the apex of the heart.

Doctor Stokes would regard the former as due to co-existent endocarditis, and the latter as the result of weakening of the heart.

Doctor Walshe conceives it possible that a basic systolic murmur may result from constriction of the roots of the aorta and pulmonary artery by exuded fibrin ; and he quotes the opinion of Martin-Solon, to the effect that a murmur of this rhythm and site may arise in the stage of serous effusion, from liquid pressure upon the roots of these vessels.

I doubt much the occurrence of murmur from either of the two last mentioned causes. I have never heard a basic murmur in the absence of valvular engagement, or of strong collateral evidence that it was of hæmic origin. On the other hand, I

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* *Dublin Journal,* vol. iv., 1834.

† *Dublin Journal of Medical and Chemical Science,* vol. vii., May 1st, 1835.

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* *Dublin Journal,* vol. iv., 1834.

† *Dublin Journal of Medical and Chemical Science,* vol. vii., May 1st, 1835.

have repeatedly found the roots of the great vessels enveloped in organized fibrin, and the pericardium distended with serum, when no such murmur existed.

I am satisfied that the cause assigned by Dr. Stokes for temporary apex-murmur, occurring in this connexion, is correct. I more than doubt the alleged resolution of valvular inflammation, in a degree sufficient to restore the normal character of the sounds of the heart, where murmur due to inflammatory exudation had previously existed; and I have already expressed the opinion, supported by clinical evidence, that initial regurgitant murmur may result from simple debility and yielding of the walls of the left ventricle, under the centrifugal pressure of the contained blood. Such a condition of the heart, involving no structural disease, would be, of course, eminently curable; and the murmur arising from it, of temporary duration, and readily suppressed by tonic treatment.

CHAPTER IV.

CAUSES OF DISEASE OF THE HEART.

THE *causes* of disease of the heart, including that of its fibrous envelope, are manifold, but may be conveniently classified under three heads; namely, hæmic, mechanical, and nutritive.

HÆMIC CAUSES ...
- Rheumatism.
- Chronic renal disease.
- Scarlatina.
- Measles.
- Small-Pox.
- Septicæmia.
- Puerperal fever.
- Phlebitis.
- Scurvy.
- Purpura.
- Cancer.
- Tuberculosis.
- Syphilis.

MECHANICAL CAUSES
- Blows or falls upon the chest ; or wounds of the pericardium or heart.
- Constriction of the chest, with laborious exercise.
- Malformation or deformity of the chest, with pressure upon the heart.

NUTRITIVE CAUSES are those which, by inducing malnutrition, lead to structural degeneration of the heart.

Hæmic causes of disease of the heart are such as operate through the blood. They consist in the presence of certain irritant principles in the circulation; whether primarily introduced from without, as in the case of the exanthematous poisons; the result of imperfect assimilation, as lactic acid; of failure of elimination, as urea; or defect of constitution, as in scurvy. These causes operate, in the first instance, as chemical irritants, and, secondarily, through the changes so produced, as mechanical

excitants of hypernutrition, with the single exception of renal
disease, the influence of which upon the heart as a morbific agent,
would seem to be exclusively mechanical.

The proclivity of persons suffering from rheumatism, espe-
cially in the acute form, to inflammatory affections of the
heart, has been known from the earliest period of medicine.
Doctor Latham* states that two-thirds of the cases of acute
rheumatism treated by him in St. Bartholomew's Hospital,
during the five years from 1836 to 1840, both included, suffered
likewise from inflammatory affections of the heart.

This is certainly in excess of the usual proportion, according
to the experience of others ; and Dr Latham, whilst admitting
that such is the case, suggests as an explanation the great assi-
duity of his clinical clerks in providing " interesting cases " for
his wards.   Doctor Sibson† records thirty-four cases of acute
cardiac affection occurring in seventy-four of acute and subacute
rheumatism ; i.e , very nearly one-half of the total number.   This
would approximately represent the proportion in which rheu-
matism has been complicated by cardiac disease in my own
experience.   The form which this complication is prone to
assume is that of inflammation of the lining or of the investing
membrane of the heart ; namely, endocarditis, or pericarditis.
Of the sum total of cardiac complications of rheumatism re-
corded by Latham, three-fourths were endocarditis, one-twelfth
pericarditis, and one-seventh consisted of the two-fold affection
Of Sibson's cases, four-fifths were endocarditis, one-tenth, peri-
carditis, and one-tenth, peri- and endocarditis combined.

As regards sex and age. In Latham's returns the proportion of
males and females affected with cardiac complication was about
equal ; and Sibson states that nearly one-half of his cases (32 out
of 74) were under twenty years of age.

Returns made for childhood (under twelve years) would, I
have no doubt, show a still larger proportion of rheumatic cases
complicated with acute inflammation of the heart ; indeed, the
occurrence of acute rheumatism without this complication in
childhood is the exception.

* *Diseases of the Heart*, vol. i , 1845, p. 144.
† *British Medical Journal*, August 13th, 1870.

Doctors Rilliet and Barthez* report that, of the children under their care affected with rheumatism, more than one-third suffered from pericarditis : namely, 4 in 11.

The following statistics, borrowed from the able and exhaustive treatise of Dr. Fuller,† are of especial value in relation to this portion of our subject. It will be seen that they deal, not only with the gross proportion of cardiac disease of rheumatic origin, but likewise with the relative prevalence of its different forms, as influenced by age and sex, in acute, subacute, and chronic rheumatism respectively.

ABSTRACT OF CASES OF ACUTE AND SUBACUTE RHEUMATISM, ADMITTED INTO ST. GEORGE'S HOSPITAL, BETWEEN THE 1ST OF JANUARY, 1845, AND THE 1ST OF MAY, 1848.

Age of Patients.	Cases of Acute and Subacute Rheumatism.	Total Heart Disease.	Pericarditis.	Endo-pericarditis.	Recent Endocardial Affection.	Old-standing Valvular, and perhaps Exo-cardial Disease.	Valvular Disease of uncertain Date.
Under 15	22	12	3	5	3	—	1
15 to 20	82	49	4	11	26	4	4
20—25	92	49	3	6	26	9	5
25—30	79	41	1	4	22	9	5
30—35	40	14	1	1	4	6	2
35—40	28	12	—	1	4	6	1
40—45	15	5	1	—	2	2	—
45—50	14	4	—	—	2	2	—
50—55	3	—	—	—	—	—	—
55—60	4	1	—	—	—	1	—
	379	187	13	28	89	39	18

Of the 13 cases of pericarditis, 6 occurred in men, and 7 in women ; or one in every 12·4 men, and one in every 6·7 women.

Endocardial affection 89, viz., 48 in men, or one in every 3·4, and 41 in women, or one in every 2·6.

* *Traité Clinique et Pratique des Maladies des Enfants*, vol. i., p. 210, quoted by Dr. Fuller.

† *On Rheumatism, Rheumatic Gout, and Sciatica*, third edition, 1860, c. ix., p. 257 *et sequent*.

ABSTRACT OF CASES OF ACUTE RHEUMATISM ADMITTED INTO ST. GEORGE'S HOSPITAL, DURING THE SAME PERIOD.

Age of Patients.	Cases of Acute Rheumatism.	Total Heart Disease.	Pericarditis.	Endo-pericarditis.	Recent Endocardial Affection.	Old-standing Valvular, and perhaps Exo-cardial Disease.	Valvular Disease of uncertain Date.
Under 15	15	9	2	5	▮	—	—
15—20	53	37	4	10	21	1	4
20—25	63	39	3	6	22	5	—
25—30	50	33	1	4	20	4	4
30—35	22	11	1	1	4	4	1
35—40	17	8	—	1	3	3	1
40—45	9	4	1	—	1	2	—
45—50	9	▮	—	—	2	1	—
50—55	1	—	—	—	—	—	—
55—60	2	1	—	—	—	1	—
	246	145	12	27	75	21	10

Of the 12 cases of pericarditis, 5 occurred in men, or in the proportion of one in 7·7 ; and 7 in women, or one in 5·2.

Of recent endocardial affection, 39 occurred in men, or one in every 2·4 ; and 36 in women, or one in every 2·1.

ABSTRACT OF CASES OF SUBACUTE RHEUMATISM ADMITTED INTO ST. GEORGE'S HOSPITAL, DURING THE ABOVE MENTIONED PERIOD.

Age of Patients.	Cases of Subacute Rheumatism.	Total Heart Disease.	Pericarditis.	Endo-pericarditis.	Recent Endocardial Affection.	Old-standing Valvular, and perhaps Exo-cardial Disease.	Valvular Disease of uncertain Date.
Under 15	7	3	▮	—	▮	—	1
15 to 20	24	11	—	1	5	3	3
20—25	29	10	—	—	4	4	2
25—30	29	8	—	—	2	5	1
30—35	18	3	—	—	—	2	1
35—40	11	4	—	—	1	3	—
40—45	6	1	—	—	1	—	—
45—50	5	1	—	—	—	1	—
50—55	2	—	—	—	—	—	—
55—60	2	—	—	—	—	—	—
	133	41	1	1	14	18	8

Of the 14 cases of recent endocardial affection, 9 occurred in men, or one in every 8·3, and 5 in women, or one in 5·1.

Pericarditis occurred in one out of every 92 men, and in one of every 41 women; or in little more than one per cent. among males, and one-half per cent. among females.

Thus it would appear that about one-half the cases of acute and subacute rheumatism registered, presented some form of cardiac affection, or 1 in every 2·02; and some form of recent cardiac affection in more than one-third of the total number, or 1 in every 2·7; and finally, some cardiac affection of long standing in more than one out of every eight cases, or 1 in 7·9.

Of the total of 130 cases of recent cardiac affection, 114 occurred in persons affected with acute rheumatism; and of those affected, nearly one-half, or 1 in every 2·06, exhibited recent heart-disease of some form.

Cardiac affection, in one of its many forms, occurred in about one-third (1 in every 3·1) of the cases of subacute rheumatism, and *recent* cardiac affection of some kind in about one out of every six and a-half cases, or 1 in every 6·6.

In the following table the preceding results are compared with those obtained by other observers.

	Total of recent Cardiac Affection.	Cases of Acute Rheumatism.	Proportion in which recent Affection of the Heart occurred.
Cases in St. George's Hospital	119	246	1 in every 2·06
Dr. William Budd's Cases ..	21	43	1 „ 2·04
M. Bouillaud's Cases ...	65	114	1 „ 1·75
Dr. Latham's Cases ...	74	136	1 „ 1·83
Dr. John Taylor's Cases ...	27·5	49	1 „ 1·78
Total ...	306·5	588	1 „ 1·91

With regard to the numerical proportion of cases of acute rheumatism complicated with pericarditis and endocarditis respectively, Doctor Fuller states that the former occurred in about one-sixth, and the latter, including the twofold affection, endo-pericarditis, in one out of every 2·3, of the cases collected by him.

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The following Tables, borrowed from Dr. Fuller's work, exhibit in a connected form the absolute and the relative proportions of the two forms of cardiac affection in acute rheumatism, according to the experience of different observers.

PERICARDITIS.

	Pericarditis.	Acute Rheumatism.	Proportion in which Pericarditis occurred.
Cases treated in St. George's Hospital	39	246	1 in every 6·3
Dr. Basham's Cases	14	66	1 ,, 4·7
Dr. William Budd's Cases	5	43	1 ,, 8·6
Dr. Latham's Cases	22	136	1 ,, 6·1
Dr. Macleod's Cases	54	307	1 ,, 5·7
Dr. Taylor's Cases	8	49	1 ,, 6·1
Total	142	847	1 ,, 5·97

ENDOCARDITIS.

	Endocarditis.	Acute Rheumatism.	Proportion in which Endocarditis occurred.
Cases treated in St. George's Hospital	107 0	246	1 in every 2·29
Dr. W. Budd's Cases	17·3	43	1 ,, 2·48
Dr. Latham's Cases	65·0	136	1 ,, 2·09
Dr. Taylor's Cases	25·4	49	1 ,, 1·92
Total	214·7	474	1 ,, 2·25

RELATIVE FREQUENCY OF RECENT EXOCARDIAL AND ENDOCARDIAL AFFECTION.

	Cases of Pericarditis.	Cases of Endocarditis.	Relative proportion of Pericarditis and Endo-carditis.
Cases treated in St. George's Hospital ...	89	107·0	1 to every 2·7
Dr. William Budd's Cases ...	5	17·3	1 „ 8·4
Dr. Latham's Cases ...	22	65·0	1 „ 2·9
Dr. Taylor's Cases ...	8	25·4	1 „ . 8·1
Total ...	74	214·7	1 „ 2·9

Doctor Fuller remarks : " The great excess of endocardial affection exhibited in these tables, arises, I believe, not from the greater frequency of endocarditis, but from the large number of cases in which a murmur is occasioned either by purely functional causes, or by temporary imperfect closure of the mitral orifice consequent on irregular contraction of the structures connected with the valves, or by the presence of fibrin deposited on the valves, without the concurrence of endocardial inflammation. I believe somewhat less than one-third of all recent cardiac murmurs met with among patients suffering from acute rheumatism will be found to result from pericarditis, and somewhat more than one-third from endocarditis,. whilst the remainder will be referable to one of the three causes above specified as contributing to the production of valvular murmur." He adds, that out of 107 cases in which endocardial murmur existed, 23 left hospital quite free from it.

Bamberger, as quoted by Niemeyer,* states that endocarditis occurs in acute articular rheumatism in the proportion of about twenty per cent. ; pericarditis fourteen per cent. ; myocarditis in much smaller proportion; and that the tendency to inflammation of the heart and pericardium is greater in proportion to the number of joints engaged.

I believe endocardial murmur may, and frequently does, arise from the first and the last mentioned cause in the course of acute rheumatism ; but I have never met with any satisfactory

* Text-Book of Practical Medicine, 1869, vol. ii., p. 481.

evidence, either clinical or necroscopic, in support of the doctrine of systolic patency of the auriculo-ventricular orifices from irregular or spasmodic contraction of the papillary muscles. I believe, further, that by far the greater number of these evanescent murmurs are due to temporary weakness or atony of the ventricular walls.

Age is a potent element in the causation of rheumatic inflammation of the heart. As shown in the preceding tables, 114 out of 130 examples of recent cardiac affection occurred in persons under 30 years of age ; and of 41 examples of pericarditis, 23 occurred in persons under the age of 20, and 37 in patients under the age of 30, leaving 4 only to be apportioned amongst those of all ages above 30. Thus it would seem that rheumatic pericarditis is essentially a disease of early life.

In acute rheumatism I have found pericarditis to exceed endocarditis in frequency ; and where the latter existed, the former was likewise present with very few exceptions. Males have been more frequently affected with cardiac complication of rheumatism than females, and the period of accession of the cardiac affection varied from the second to the twenty-seventh day, from the date of first illness. The friction-sound has been of every conceivable rhythm and site; from double systolic (case of Mary N.) at base, to prediastolic at apex (case of Mary K). Presystolic friction-sound at apex I have repeatedly met with (see cases of L. O'B. and J. D.) ; it is interesting chiefly with reference to differential diagnosis from the presystolic murmur of mitral constriction. The complications of rheumatic pericarditis which I have met with, have been pleuritis (single and double), myocarditis, and endocarditis.

The murmur of acute rheumatic endocarditis is, with very few exceptions, mitral as to origin, and systolic as to rhythm. Once established it is usually coeval with the life of the patient. My experience, in regard to this matter, does not by any means coincide with that of Dr. Sibson, who discharged one-half of his cases of endocarditis " well and free from cardiac valve-murmur ;"* nor with that of Dr. Latham, seventeen of whose cases, out of a total of sixty-three, or nearly one-fourth, left hospital

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* *British Medical Journal*, August 13th, 1870.

free from murmur.*   I have not had the good fortune to meet
with more than one such case out of some hundreds; that is,
complete cure of endocarditis, in the sense of final cessation
of a previously well pronounced endocardial murmur; which, as
regards endocarditis, is the *only* evidence of cure I am willing to
admit.

Doctor Sibson has correctly stated† that the earliest announce-
ment of valvular engagement in acute endocarditis, consists in
prolongation of the first sound (cases of Jane A. and Mary N.),
which, in the course of a few days, is gradually transformed into
veritable murmur.   I have had, however, no experience of the
inverted order of these phenomena which has so often fallen
under the cognizance of Dr Sibson; namely, the transition of
murmur into prolonged sound, as a preliminary step to its final
extinction.

The portion of the civil population most liable to cardiac disease
are precisely those who are most exposed to the causes of rheuma-
tism, and, pre-eminently, to sudden changes of temperature with
insufficient clothing, depressed spirits, and deteriorated health.
Tailors and milliners, especially, are examples of the truth of
these remarks.   After long confinement in their miserable work-
shops, overheated by gas, ill-ventilated, and with insufficient
cubic space for the numbers crowded into them, these poor
people are exposed to cold in travelling to, or actually whilst
resident in, their humble and ill-provided lodgings.   Their food
is unwholesome or insufficient, their clothing light, and their
minds depressed by the unsuccessful struggle for existence.
Rheumatism, of a very acute character, and complicated with
cardiac inflammation in a proportion higher than the average,
is consequently of common occurrence amongst them.

The agent by which, in rheumatism, valvular inflammation is
produced, is generally considered to be lactic acid.   Dr. Richard-
son has shown, experimentally,‡ that this agent introduced within
the peritoneum of a living animal, is capable of exciting acute
inflammation of the endocardium, manifested chiefly upon the
valves.

* *Opus citat.*, p. 147.   † *Loco citat.*   ‡ *The Asclepiad.*

Doctor Stokes remarks,* with truth, that cardiac and articular inflammation are, alike, only accidents of the rheumatic fever; and that either, or both at the same time, may complicate it. He mentions two examples of rheumatic pericarditis, in one of which, observed by himself, the symptoms of pericardial inflammation preceded the implication of the joints by a period of ten days; and in the other, which occurred in the practice of Dr. Graves and was declared by symptoms and signs, the interval preceding arthritis was five days   In reference to rheumatic pericarditis he has arrived at the following conclusions; viz.,

1. "That though the combination of pericarditis is common, yet that the disease of the heart is more closely related to the rheumatic fever than to the inflammation of the joints.

2. "That the liability to pericarditis is in direct proportion to the violence and duration of the fever.

3. "That in the apyrexial cases of acute arthritis, the liability to cardiac inflammation is but slight.

4. "That pericarditis may be developed at any period of the disease, and even precede the arthritis.

5. "That every variety and degree of pericarditis may occur in connexion with acute rheumatism, from the simple, dry, latent pericarditis, to the worst forms, combined with inflammation of the endocardium and muscular structure."

He notices three forms of rheumatic pericarditis: viz., 1st, that in which the disease, as regards symptoms, is latent, physical signs alone declaring its existence; 2ndly, that which is announced by a train of symptoms recently set up, and dependent upon copious liquid effusion; and, 3rdly, that in which symptoms of cardiac distress precede the physical signs by one or two days.

As regards the relative frequency of acute inflammation of the pericardium and endocardium, single and combined, he has come to the conclusion that it may be represented by the following series:

1. Acute pericarditis, with endocarditis.
2. Acute pericarditis, without endocarditis.
3. Endocarditis, without pericarditis.

* *The Diseases of the Heart and Aorta*, 1854, p. 46, *et sequent.*

Guided by what I have personally witnessed, I believe that the foregoing scale correctly represents the relative numerical proportion in which acute pericarditis and endocarditis are met with in actual practice.

There are several sources of fallacy which, if not carefully avoided, may lead to an opposite conclusion. These will be found fully discussed in connexion with the subject of endocarditis. Here it is only necessary to state that, in my opinion, acute primary pericarditis is of more frequent occurrence than acute primary endocarditis. I do not, however, coincide with Dr. Stokes in the opinion that acute pericarditis is more frequently met with in combination with acute endocarditis, than in the isolated form. Indeed, I hold that the compound disease should be placed, not at the head, but at the foot of the scale.

The occurrence of hypertrophy of the heart, as a sequel of chronic renal disease, was first noticed by Dr. Bright. After narrating one out of many cases illustrative of this connexion, he remarks : " What influence the state of the heart exerts over the kidney is less obvious, nor is it clear that it is at all instrumental in promoting the mottling (granular disease) of the organ, though in many cases the two organs are found simultaneously diseased."*

From this extract it is evident that Bright, even at this early date, had perceived a relationship between renal and cardiac disease, although he did not rightly interpret the connexion. Subsequently, however, he exhibited a clearer insight into the mutual relationship of these two diseases. After pointing out the connexion of hypertrophy of the left ventricle with renal disease, he says: " The obvious structural changes in the heart have consisted chiefly of hypertrophy with or without valvular disease, and what is most striking, out of fifty-two cases of hypertrophy, no valvular disease whatever could be detected in thirty-four; still, however, leaving twenty-two without any probable organic cause for the marked hypertrophy generally affecting the left ventricle." In explanation of the pathological relationship he assumes either a direct irritant action of the blood upon the heart, or that it " so affects the minute and

* *Reports of Medical Cases*, 1827, vol. ii., part i., p. 240.

capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system."[*] Thus, to Bright is clearly due, not only the discovery of the connexion between left ventricular hypertrophy and chronic renal disease, but likewise that of the causative relationship in which the latter stands towards the former affection, and the correct theory of its operation, namely, minute arterial and capillary resistance.

Doctor George Johnson supplemented and confirmed this theory,[†] by showing that in all the forms of chronic renal affection included under the designation of Bright's disease, the muscular coats of the minute renal arteries are hypertrophied ; and further, that hypertrophy of the systemic arteries exists in these diseases.[‡]   Finally, he offers a connected view of his theory in a serial form as follows ; viz., blood contamination by retained excreta, capillary resistance, hypertrophy of the minute arteries, and hypertrophy of the left ventricle.[§]

Jaccoud[‖] accepts the mechanical theory of Traube in explanation of hypertrophy of the heart in Bright's disease generally, but he does not consider it of universal application.   In many cases he regards it, with Bright, as due to the altered state of the blood.

According to Dr. Grainger Stewart,[¶] cardiac hypertrophy was present in 40 per cent. of his cases of the " inflammatory form " of Bright's disease, and was apparently referable solely to the kidney disease.   The proportion in which hypertrophy existed increased with the stage of the disease thus :—

1st stage, 12 per cent. of the fatal cases.

2nd stage, 38 per cent.

3rd stage, 100 per cent.

In the "amyloid" form he met with cardiac hypertrophy only in the proportion of 4 per cent. of the cases, and in all of these the renal disease was in its third or atrophic stage.

In the "cirrhotic" form of the disease 46 per cent. of the cases

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* *Guy's Hospital Reports*, vol. i., 1836.

† *Med. Chirurg. Transactions*, vol. xxxii.

‡ *Lancet*, December 21st, 1867.

§ Clinical Lecture, *British Medical Journal*, March 7th, 1868.

‖ Clinical Lectures on Medicine, delivered at the Charité Hospital, Paris, 1867.

¶ *Bright's Disease*, 1868.

presented enlargement of the heart, which was attributable to the renal disease exclusively; whilst the proportion in which cardiac hypertrophy, with valvular or vascular disease, was associated with cirrhotic disease of the kidneys, was still larger.

Amongst the hæmic causes of disease of the heart I regard the poison of scarlatina as next to rheumatism in frequency, and perhaps superior to it in potency. It attacks the endocardium by preference, seldom engaging the pericardium, and more frequently the mitral than the aortic valve.

Many of the most formidable examples of valvular lesion that I have met with owed their origin to scarlatina. The patients are generally children, and rarely survive the second period of life. The complication is usually declared in the second week of the fever, but occasionally in the first week, or in the stage of desquamation.

Trousseau maintains that the frequency of cardiac complication in scarlatina is due, not to that disease, but to the coexistence of rheumatism, which, as he alleges, is a not unusual concomitant of scarlatina, occurring in the proportion of one-third of the cases in adults, and manifested by pains in the joints from the third to the eighth day. Under these circumstances, endo- and pericarditis frequently occur in the stage of decline of the scarlatina, the former somewhat more frequently than the latter.*

In the course of measles and small-pox likewise, but less frequently than in scarlatina, from the operation of the specific poison of these diseases, endocarditis is set up, and followed, in time, by the usual structural alterations of the heart. In the low, asthenic, or typhoid form of small-pox, however, pericarditis, latent, or declared only by physical signs, is the usual cardiac complication. Such is likewise the case in diffuse inflammation, puerperal fever, and phlebitis, as long since pointed out by Dr. Stokes.

Scorbutus and purpura occasionally give rise to pericarditis of the hæmorrhagic form, or may complicate an ordinary pericarditis by determining extravasation of blood into the pericardium.

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* *Clinical Medicine*, Sydenham Society's edition, 1868, vol. i., p. 397.

This subject will be discussed further on in connexion with that of pericarditis.

Cancerous and tuberculous pericarditis are also of occasional, but very rare, occurrence.

Ricord describes morbid changes of the heart and other organs, which he compares to syphilitic tumors or nodes.*

Amongst the *mechanical* causes of disease of the heart, blows or falls upon the chest hold a prominent place; they usually act injuriously upon the organ by causing rupture of the chordæ tendineæ or semilunar valves, or detachment of the latter from the walls of the vessels.

Wounds of the heart are very rarely the cause of chronic disease or functional impairment of the organ; they are usually followed either by proximate death, or ultimate and complete recovery. Extravasation of blood into the pericardium, consequent upon a wound, may, however, give rise to pericarditis and its results.

Constriction of the chest, as by tight lacing or bracing, especially if conjoined with laborious exercise, is eminently conducive to cardiac hypertrophy, and disease of the arch of the aorta, from extra tension. It is in this way that the excessive mortality from disease of the heart and aorta in the British army is to be accounted for.

Doctor Robertson has shown† for the four years ending 1866, a mean ratio of mortality from disease of the heart of 40 per 1,000 in the army, and 12 per 1,000 in the navy. This formidable mortality in the army from cardiac disease, he attributes, in an especial manner, to the severe drill to which the young recruit is subjected whilst tightly braced up in a close-fitting jacket and high stock. The "running drill," in particular, he regards as most pernicious in its operation; and he sustains this opinion by reference to the number of admissions into hospital for cardiac disease amongst the Royal Marine Artillery, during the two years preceding, and those immediately succeeding, the introduction of that form of drill into the corps. In the year

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* *Clinique Iconograph. de l'Hôpital des Vénériens.*

† "On Diseases of the Heart and Aneurism in the Royal Navy and Army," by G. Robertson, M.D., *Medical Press and Circular*, July 7th, 1869.

1865 the number was 24, and in 1866 it was 26. In the summer of 1867 the running drill was introduced, and the admissions for cardiac disease the year after its introduction amounted to 47, in an average force of 1,750 men. During the first quarter of the succeeding year the number was 25. This drill is practised only during the winter months; and despite the severe drill this body of men undergo habitually in the summer, he observes that during that season the number of admissions for disease of the heart has been much below that registered in the winter.

The proportionate excess of cardiac disease in the navy, Dr. Robertson inclines to attribute to rheumatism, generated by the twofold cause of vicissitudes of temperature and the constant habit of washing the decks.

Assistant-Surgeon Myers* treats the subject of excessive prevalence of cardiac disease in the army with much ability; and, with the view of exhibiting this in contrast with the ratio amongst corresponding classes of the civil population, he shows the proportion of rejections for disease of the heart amongst recruits for the years 1864, '65 and '66, respectively, to have been 19·78, 22·22, and 25·13 per 1,000.

Inspector-General Lawson, as quoted by him, gives the general mortality from disease of the circulating system, including aneurism and cardiac disease, in the civil population and the army, respectively, between the ages of 15 and 44 years, as follows:

Civil population	..	..	..	·48 per 1,000
Army	...	...	...	·84 ,,

and, excluding aneurism,

Civil population	...	...	...	·45 per 1,000
Army	...	...	...	·50 ,,

But even the preceding table does not fairly represent the relative mortality; because soldiers invalided for disease of the heart are placed to the account of the civil community, and their deaths are registered against it. Thence arises a twofold

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* *Prize Essay on the Etiology and Prevalence of Diseases of the Heart among Soldiers,* 1870.

derangement, namely, reduction in the ratio of fatal cardiac disease in the army, and undue elevation of it amongst civilians.

Mr. Myers further gives, as the result of his own inquiries, the following figures, representing the relative mortality from disease of the heart amongst civilians and soldiers of corresponding ages:

	Army.	Civilians.
20 to 24 years	·12	·25
25 and upwards	1·56	·94

Thus, it would seem that within the age of 20 to 24 years the death-rate from this cause is lower in the army than amongst civilians; but from 25 years upwards it is much higher. No doubt soldiers, who are admitted into the service at or about the age of 20 *on account* of their good health, contrast favourably in this respect with an equal number of civilians who are *not* selected with reference to this condition; and at the age of 24, these youths have not been as yet subjected to the various causes, which confer upon the military service of Great Britain the distinction of a preponderating mortality from disease of the heart, for a sufficient length of time to develop their full operation.

For the purpose of exhibiting the relative death-rate and invaliding from cardiac disease in the naval and military services, Mr. Myers submits the following statistics, given in summary, for the period 1860 to 1865 inclusive:

	Died	Ratio per 1,000 of admitted.	Invalided.	Ratio per 1,000 of admitted.
Army	259	·90	1·506	5·26
Navy	228	·66	1·184	3·44

With reference to the special forms of disease of the circulatory system found to prevail in the army, the annexed Tables, borrowed from Mr. Myers's work, are of much value:

RELATIVE PROPORTION OF DEATHS AND INVALIDING IN THE HOME SERVICES, OWING TO THE VARIOUS DISEASES OF THE CIRCULATORY SYSTEM, IN THEIR ORDER OF FREQUENCY, DURING THE FIVE YEARS, 1863 TO 1867.

Diseases of the Circulatory System.	Deaths.	Ratio per 1,000 of Total Deaths.
Aneurism	138	431·23
Morbus Valvularum Cordis	103	321·87
Hypertrophia Cordis	21	65·62
Degeneratio Cordis	21	65·62
Pericarditis	21	65·62
Morbus Cordis (doubtful)	5	15·62
Carditis	4	12·5
Atrophia Cordis	3	9·37
Atheroma Arteriosum	2	6·25
Varix	1	3·12
Syncope	1	3·12
	320	...

Diseases of the Circulatory System.	Invalided.	Ratio per 1,000 of Total Invalided.
Morbus Valvularum Cordis	518	369·98
Varix	473	357·41
Hypertrophia Cordis	201	149·26
Morbus Cordis	37	27·48
Aneurism	35	26·
Carditis	29	21·54
Palpitatio	21	15·6
Pericarditis	17	12·63
Angina Pectoris	6	4·45
Atrophia Cordis	4	2·97
Syncope	3	2·22
Degeneratio Cordis	2	1·48
	1,346	...

As regards aneurism, the difference observable in the foregoing tables would seem due to difficulty of diagnosis.

But it may be said that from rheumatism, which operates so injuriously amongst the civil population as a cause of cardiac disease, the soldier is not exempt; nay, it may even be assumed that in the camp and bivouac he is more exposed to the causes of rheumatism and, its frequent consequence, organic disease of the heart, than persons of corresponding classes engaged in civil

avocations. But, exposure of this kind occurs only in the time of war, and is, therefore, exceptional. Again, there is no evidence whatever that rheumatism is more prevalent in the army than amongst the civil population; whereas morbus cordis notoriously is so.

Doctor Maclean has shown* that out of one hundred and fifty-one patients treated for heart-disease in Netley Hospital, six only had suffered from acute rheumatism.

After rheumatism, the most frequent cause of morbus cordis is renal disease; this, however, operates chiefly after the period of life when the soldier retires from active service. The same may be said of degenerative structural changes in the heart.

Amongst seventy patients suffering from cardiac disease at Netley, Dr. Parkes was able to collect the histories of only two or three who had been the subjects of rheumatic, renal, syphilitic, or other affections, of a character adequate to account for the supervention of disease of the heart.

In India, for the years 1865, 1866, and 1867, the invaliding on the score of heart-disease was only in the proportion of 3·88 per 1,000. Hence, residence in that country cannot be adduced as a special cause of the prevalence of heart-disease amongst soldiers. The excessive mortality and invaliding from this cause in the army, must, therefore, be charged to the account of some agency, to the operation of which the military service is peculiarly exposed.

Doctor Maclean says :† " The labouring men have their clothes perfectly free, so do also sportsmen and tourists ; but the soldier is compelled to do his work under the utmost possible disadvantages, as regards the weight he has to carry, the mode of carrying it, and the entire arrangement of his dress and equipment. Many men fall out in a state of extreme distress, and many surgeons assure me that nothing but a strong feeling of *esprit de corps* prevents many more from doing so."

Mr. Myers writes:‡ " In civil life, when any prolonged or violent exertion of the body has to be made, be it for pleasure or

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* *Army Medical Report,* 1867.
† *Journal of the United Service Institution.*
‡ *Opus citat.,* p. 32.

otherwise, the great point first attended to is to remove all the various constrictions of ordinary dress, it being well known by all classes of society how necessary it is, under such circumstances, to allow the chest its fullest powers of expansion, and consequently the greatest possible freedom to the heart and lungs.

"Now, the method of equipping the soldier is directly opposed to this. His tunic is made to fit as tightly as the skill of the tailor can accomplish, any defect in this respect being probably corrected, after the careful scrutiny of the colonel or adjutant, by a little paring, and finally, by the addition of a padded waistcoat, which the soldier takes upon himself to supply.

"His waist-belt adds to the constriction below the chest, and his tunic collar above it (with or without the stock), and then, to complete the artificial chest-case, the knapsack straps supply all that is requisite, whilst the pouch-belt adds its share to the general compression.

"The chest thus fixed, as it were, in a vice, has little or no power of expansion, and the circulation through heart, lungs, and great vessels is proportionately impeded."

Mr. Myers adduces the following extract from the report of the Government Committee, appointed in 1864 to inquire into this subject, as confirmatory of the opinion urged by himself in the preceding passages.

"During exertion, the movements of the chest increase greatly; deeper breathings are made, the diameter of the chest enlarges in all directions, causing greater expansion of the lungs; the blood flows much more rapidly, and the changes in it, and the evolution of carbonic acid are trebled and quadrupled in amount, and the heart acts much more quickly and forcibly. If anything destroys the equilibrium between the powerful action of the heart, and the capacity of the lungs to receive the blood propelled into them by the heart, the necessary consequence is an accumulation of blood in the cavities and walls of the heart, which leads to an imperfect action of that organ, and to organic changes in its cavities and walls." He adds: "When experiments were made at Chatham to ascertain the comparative amount of distress caused by the accoutrements of various

European armies, it was unquestionably proved that, though all produced it more or less, ours ranked first in prejudicial effect."

It would appear, as indeed might have been anticipated, that next after hypertrophy of the left ventricle, the form which organic disease of the heart most commonly assumes in the army is that affecting the aortic valves. Thus, as shown by the *post mortem* returns of Netley Hospital from October, 1860, to November, 1869, the numerical proportion of the several valvular lesions stood thus; viz., aortic valve disease, 50; mitral valve disease, 25 ; aortic and mitral valve disease, 22.*

Doctor Maclean's returns show a less striking disproportion; thus: aortic valve disease, 72; mitral valve disease, 54; total cases, 126.†

Barclay, Chambers, and Ormerod show the following relative proportion between these several forms of valvular lesion in civil life; viz.: aortic valve disease, 192 ; mitral valve disease, 205; aortic and mitral valve disease, 259 ; total cases, 673.‡

Inspector-General Lawson showed that deaths from aortic aneurism were eleven times more numerous in the army than in the male civil population ;§ and Dr. Maclean states: " Between April 1st, 1867, and April 1st, 1869, thirty-six cases of aneurism of the aorta were under treatment in the clinical wards of Netley," and in these there was evidence of " a rheumatic history in five, and a distinct syphilitic history in only three of the cases."∥

The excessive use of alcohol has been charged with the preponderance of aneurism in the army; but on apparently insufficient evidence.

The difficulties opposed to the circulation of the blood through the arch of the aorta by the constriction of the chest, due to the soldier's dress, would seem to afford a more rational explanation. Syphilis, though, unfortunately, very prevalent in the army, is likewise common amongst the civil population of towns; and the difference in its relative prevalence in the two classes men-

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* *Opus citat.*, p. 57.

† *Army Medical Report*, 1867.

‡ *British and Foreign Med. Chirurg. Review*, vol. xiv.

§ *Army Medical Report*, 1866.

∥ *Ibid.*, 1867.

tioned, is by no means proportionate to that which represents the disparity in the per centage of aneurism amongst them. I believe the conclusion is inevitable, that the greater number of these cases is due " to mechanical obstruction to the circulation in the soldier when he is undergoing exertion, caused by the general constriction of his neck and chest by faulty clothing and accoutrements."* If this conclusion be legitimate, it must follow that, in the soldier, the arch of the aorta is most frequently the seat of aneurism; and that such is actually the case the following statistics adduced by Mr. Myers would seem to prove: ascending aorta, 37; arch, 38; descending, 12; thoracic, 7; abdominal, 15; total number of cases of aortic aneurism, 109.†

It behoves every humane man who has been made sensible, by the evidence of the statistics now adduced, of the great and preventable sacrifice of life imposed upon the rank and file of the British army by an error of the War Office, to raise his voice against it, and to urge the officials at the head of this department to endeavour, at length, to comprehend the elements of a problem which everybody who has thought over the matter, except themselves, would seem to have long since mastered. This official blindness seems the more unaccountable, since the information by which the public mind has been enlightened on this subject has been supplied by the Army Medical Reports exclusively. The independence and public spirit exhibited by the medical officers by whom these reports have been compiled, is most creditable to the Army Medical Service, and cannot fail to be ultimately productive of reform in the dress and accoutrements of the soldier.‡

Deformity of the chest, more especially that known as "pigeon-chest," by interfering with the movements of the heart, becomes a cause of mechanical irritation, and, ultimately, of hypertrophy of the organ. Incurvation of the spine, whether antero-posterior or lateral, by inducing contraction of the chest in one or more of its diameters, operates in a similar manner.

* Mr. Myers, *Opus citat.*, p. 70.

† *Opus citat.*, p. 72.

‡ Since the above was written we are glad to learn that certain improvements have been made in this respect.

Hence, in all such cases the action of the heart is quick and tumultuous. At first the sounds are sharp and clear, as in simple functional excitement ; but, after some time they become masked, owing to the supervention of hypertrophy from persistent and excessive functional activity.

In a similar manner, hypertrophy of the heart may be, and frequently is, induced by artificial constriction of the chest, as exemplified in the vicious habit of tight lacing. Than this habit, so prevalent amongst young females of the affluent classes, no example can be adduced more strikingly illustrative of the practical evils arising from ignorance of the elements of physiology. If ladies were instructed in the first principles of animal mechanics, the laws which govern respiration and circulation, and the penalties attached to their infringement, it is not likely, despite the inexorable requirements of fashionable society, that they would wilfully provoke the terrible consequences of early disease of the heart.

Diseases of degeneration of the heart are due to two several causes ; namely, direct failure of nutrition, and interstitial exudation. To the former cause are attributable softening, attenuation and fatty degeneration of the organ, and also calcification of adventitious deposits in or upon it; and to the latter, congestive hypertrophy, induration, and atrophy of tissue.

CHAPTER V.

DISEASES OF THE HEART.

DISEASES of the heart are divisible into two great classes; viz.,
1. Organic.
2. Functional.
>   *Organic* diseases comprise four groups; namely, those
>   engaging :
>   The investing membranes,
>   The parietes,
>   The valves, and
>   The nutrient vessels of the heart.
>   *Functional* diseases of the heart are of two kinds ; viz.,
>   Muscular asthenia, and
>   Neurosis.

Pericarditis, or inflammation of the pericardium, is presented
under one of two forms; namely, the acute and the chronic.

Acute pericarditis is usually an accompaniment or a compli-
cation of acute or subacute rheumatism ; it may, however, be
the only well pronounced rheumatic manifestation. The disease
is most frequently declared in the course, and within the first
week, of an attack of articular rheumatism. Usually the articular
pain is either greatly mitigated, or even entirely suspended, on
the supervention of pericarditis. Acute pericarditis may, how-
ever, be the result of wounds or other mechanical injuries of the
pericardium, of the irruption of pus, as from an hepatic abscess ;
or of the exudation or leakage of blood into the pericardium, as
in the hypinotic state. It may likewise arise as a consequence
of ulceration of the heart or pericardium ; from exposure of the
body, whilst heated, to the influence of cold; from retrocession of
gout or erysipelas, as stated by Mr. Adams ; or in the course of
an exanthem. Phlebitis, or pyæmia, and the rapid cure of pso-
riasis by local applications (Wood), have been likewise adduced
as causes of acute pericarditis.

Fuller states that it complicates rheumatism under ordinary treatment once in 5·97 cases.* He has met with six cases in which pericarditis occurred from two to seven days before articular inflammation, and he holds that these two affections exercise no reciprocal influence, the one upon the other. I cannot help thinking, however, that in this opinion he is mistaken. I rather agree with Dr. Sibson,† who maintains that inflammation of the joints does exercise a very potent influence, by reflex irritation, in determining the occurrence of pericarditis.

Three stages of the disease have been usually distinguished, as clearly stated by Dr Mayne;‡ viz, 1st, that of suspended secretion of serum, and consequent dryness of the pericardium, the only pathological change, with the exception of its opposite, serous effusion, recognized by Collin;§ 2nd, effusion of lymph or fibrin; and, 3rd, effusion of serum.

Doctor Mayne was of opinion that the first of these stages was declared only by general symptoms; viz, oppression of breathing, rapid pulse (130 to 140), anxious expression of face, etc. He stated that, in his experience, it repeatedly preceded friction-sound by thirty hours, as inferred from the existence, during this period, of the symptoms above mentioned.

Graves states that excited and irregular action of the heart often *precedes* the signs of pericarditis; and Stokes has observed the symptoms of pericarditis to precede friction-sound by two days.

Mayne was of opinion that the pericardium in the state of simple dryness of its surface, is incapable of yielding an audible attrition-sound.

Collin, on the contrary, held that this is actually the condition of the pericardium indicated by the *bruit de cuir neuf*

Stokes doubts, but does not deny, the competency of simple dryness of the pericardium to produce *frottement*.‖

* *Diseases of the Chest*, 1862, p. 153, *et sequent*
† Address in Medicine. British Medical Association. *Pamphlet*, 1870, p. 3.
‡ *Dublin Journal of Medical and Chemical Science*, vol. vii, May 1st, 1885
§ *Commentaires des Propositions de Pathologie*, 1829, tom. i, p. 399
‖ *Dublin Journal of Medical and Chemical Science*, vol iv, September 1st, 1833.
In his work (*Diseases of the Heart and Aorta*, p. 9) published more than twenty years later (1854), Dr. Stokes admits, with scarcely any qualification, the occurrenc

Doctor Walshe holds that a state of dryness, with vascular engorgement of the free surface of the pericardium, is competent to yield friction-sound of a grazing, or even of a scraping character, during the movements of the heart.*

I have never met with a case which would warrant me in asserting, that a state of simple dryness and vascularity of surface may give rise in the pericardium to veritable friction-sound. I do not, however, deny the possibility of an occurrence, which, theoretically, would seem not improbable. In every instance, without exception, in which I have had the advantage of determining, by *post mortem* examination of the body, the condition of the serous surface of the pericardium, where friction-sound of indubitable pericardial origin had existed during the patient's last illness, I have found lymph in greater or less quantity effused upon that surface; and out of a great number of such examples, I cannot recall one which afforded the slightest warrant for the assumption that the lymph so effused was in process of removal by absorption, notwithstanding the positive assertion to this effect made by many writers of eminence.

If it is possible, as Dr. Mayne held,† and in this opinion I quite coincide with him, to predict from general and special symptoms the proximate advent of pericardial *frottement*, the pericardium must exhibit, concurrently with the manifestation of these symptoms, the earliest pathological change which characterizes a serous surface in process of inflammation, namely, capillary injection and suspension of secretion. In two of Dr. Mayne's cases the diagnosis of pericarditis was made from symptoms alone, thirty hours before *frottement* was perceptible.

I have repeatedly, in the course of rheumatic fever, anticipated the advent of pericardial *frottement*, by at least twenty-four hours, from the sense of precordial oppression experienced by the patient, the accelerated breathing and pulse, anxious

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of pericardial *frottement* in the first stage of pericarditis, from simple dryness of the serous surface; and Dr. Hope (*opus citat.*, p. 168) thinks that "deficient lubricity of the pericardium, from defective secretion in the earliest stage of inflammation, may possibly be *one* of the causes of the creaking sound, independent of lymph."

* *A Practical Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1862, p. 216.

† *Loco citat.*

expression of countenance, and, especially, from the abrupt,
energetic, and tumultuous action of the heart, associated with a
peculiar rustling sound ; other acute inflammations of the chest
having been excluded. In all the above examples, I believe that
the serous surface of the pericardium was already vascular and
dry; yet, by the most careful examination, friction-sound was not
then detectable. Within the course of the succeeding twenty-
four hours, however, indubitable attrition-murmur became dis-
tinctly audible, and converted the *presumptive* into a *positive*
diagnosis of pericarditis.   Without the confirmatory evidence
afforded by friction-sound, it is manifest that a positive diag-
nosis of pericarditis could not have been made in the cases
adverted to, because the symptoms, taken individually or col-
lectively, might have been due to other causes, however strong
the probability of their dependence upon pericarditis. Da Costa
declares that pericarditis can be recognized only by means of
physical signs, adding, there are no general symptoms that prove
a pericarditis to exist, not even that of the so-called character-
istic precordial pain.* Corvisart, and even Laennec himself,
strange as it may seem, were unacquainted with the physical
signs of pericarditis , their diagnosis of this affection rested upon
presumptive evidence alone.   Bertin, Broussais, and Andral
laboured under the same disadvantage, notwithstanding that
the discovery of the pathognomonic sign of pericarditis, the *bruit
de cuir neuf*, was published by Collin the same year (1824) in
which Bertin's book appeared, but some months earlier, as shown
by the disparaging allusion to it contained in that work, two
years before the second edition of the celebrated *Traité de l'Aus-
cultation Médiate*, and five years anterior to the works of Andral
and Broussais. The latter, indeed, whilst fully admitting the
diagnostic value of the sign discovered by Collin, gives no evi-
dence that he had made much use of it in practice. He says :—
" But there is a phenomenon worthy of more attention than it
has, as yet, perhaps received. It is the sound of parchment,
which one perceives distinctly by means of the stethoscope.

" In exploring with this instrument, in cases of incipient peri-
carditis, one experiences the sensation yielded by two dry bodies,

* *Medical Diagnosis*, third edition, 1870, p. 355.

such as parchment, rubbing against one another; and this sign, when associated with pain and anguish, can leave no doubt as to the existence of inflammation. There is no reason why we should not profit by this observation for the purpose of making a precise diagnosis of the disease," etc.*

Still more singular is it that Laennec, who has made such free and profitable use of the stethoscope in the diagnosis of pulmonary and cardiac disease, should have failed to recognise its application in that of pericarditis, and not have noted the striking and pathognomonic phenomenon of friction-sound in connexion with that disease. Collin was no stranger to Laennec, as, in the preface to the second edition of his great work, the illustrious discoverer of mediate auscultation proclaims his indebtedness to that distinguished observer for much and valuable assistance rendered in the collection of his clinical records.

Doctor Latham was one of the first physicians who recognised the diagnostic value of pericardial *bruit.* He says:—" I am not called upon to determine the general value of auscultation, as an aid to diagnosis. Probably it does not deserve all the high commendation of its inventor and its early advocates, and still less the absolute contempt and rejection which it has incurred at the hands of others. I confine my remarks to a single sign derived from it, contributing something, I believe, towards the diagnosis of a particular disease, and am content to affirm, that during more than three years in which I have practised the method of auscultation with some diligence, and, as a security against self-deception, have admitted no result of my own observation which has not been confirmed by that of others, the sign in question, of the peculiar sound accompanying the contraction of the ventricles, has not been absent in any one authentic case of pericarditis. And in three years the number of such cases in so large an hospital as St. Bartholomew's is considerable. My observation is restricted to *rheumatic pericarditis.* The same sign may attend pericarditis arising under other circumstances, but I do not know that it does.

" In rheumatic pericarditis the brouissement, or bruit de souf-

* *Loco citat.*

flet, is always among the earliest symptoms referable to the heart, and sometimes the very first."*

From the preceding extract it will be seen that, whilst identifying and correctly associating the acoustic sign of pericarditis, Dr. Latham failed to appreciate its precise pathological significance, and the full extent of its diagnostic application. Nevertheless, he is entitled to distinguished notice in the history of this subject.

To Dr Stokes belongs the merit of having been the first not only to assign their full diagnostic value to the acoustic phenomena of pericarditis, but to lay down precise rules, not even now diminished in value, for their identification.†

In his valuable and original paper, six cases of pericarditis are narrated, which may be briefly summarised as follows :—

Case 1. Loud friction-sound and *frémissement*, due to a second and recent attack of pericarditis, were perceived. Old adhesions and much semi-cartilaginous deposit were found on *post mortem* examination of the body.

Case 2. Double friction-sound, but varying in quality at different points of the pericardium, and not audible beyond one and a-half inch from the precordium, whilst the normal sounds of the heart were extensively audible. *Frottement* persisted at one point in the precordial region, whilst elsewhere it had ceased. The diagnosis of adhesion of the pericardium at all points, save that just mentioned, was made, and on *post mortem* inspection of the body, was found to be correct. This was an example of "latent" pericarditis , *i.e.*, in which the presence of the disease was announced by physical signs exclusively.

Case 3. Latent and dry pericarditis. Friction-sound varied in quality in different situations. Diagnosis confirmed by autopsy.

Case 4. A child, aged five years. Double friction-sound audible under both clavicles, and along the spine. Extensive precordial dulness, and epigastric tenderness. On examination of the body after death, dry pericarditis, and considerable hypertrophy of the heart, with slight recent thickening of the mitral and aortic valves, were found.

* *London Medical Gazette*, vol. iii., January 17th, 1829, p. 314.
† *Dublin Journal of Medical and Chemical Science*, vol. iv., September 1st, 1833.

Case 5. Double friction-sound, which underwent change of quality and situation in the progress of the case, and exhibited, moreover, a respiratory rhythm. *Post mortem* examination not obtained.

Case 6. Double friction-sound which underwent change of character and site, and on the supervention of precordial dulness, ceased to be audible. Dulness subsequently cleared up, and friction-sound became again audible, and in the course of convalescence assumed the character of *bruit de soufflet.* Recovery.

These cases were of the utmost value to the science of medicine at the time they were published, because they settled affirmatively, once and for ever, the question of the practicability of diagnosing pericarditis actually in progress, by means of physical evidence exclusively. But, furthermore, from the observation of these cases, rules were constructed for the differential diagnosis of pericarditis, which may safely challenge comparison with anything since written on this subject.

In an especial manner are the variations in the intensity, quality, and rhythm of friction-sound in different situations within the precordium; and its modifications from day to day, and under treatment, insisted upon, as distinctive of this phenomenon.

In the progress of this work I shall have occasion again to refer to this important contribution to medical literature.

Doctor (now Sir Thomas) Watson followed, at a short interval, with a valuable paper* confirmatory of Dr. Stokes' opinion as to the unique character and special diagnostic value of attrition-sound in pericarditis; contributing cases in illustration, one of which, as an example of latent pericarditis, was of especial value at this period, when the possibility of the diagnosis of that disease from acoustic and tactile evidence exclusively was still called in question. The diagnosis was confirmed by *post mortem* examination of the body.

In the same year Dr. Mayne contributed a memoir of the utmost value on this subject.† His cases, which are numerous,

* *London Medical Gazette,* vol. xvi., part i., April 11th, 1835.
† *Dublin Journal of Medical and Chemical Science,* vol. vii., May 1st, 1835.

would alone be sufficient to establish the independent physical diagnosis of pericarditis.   Yet, he declares it as his opinion, that physical signs are not of themselves sufficient in all cases; adding, "In some cases this sign (friction-sound) will not be evident at any one period of the entire disease, and in others it may readily be mistaken for similar sounds which are characteristic of other organic lesions."   The differential diagnosis from endocarditis is, he says, in many instances attended with the greatest difficulty; especially is this the case when pericarditis has supervened upon organic disease, the stethoscopic phenomena being due exclusively to the latter.

I cannot agree in the opinion that pericarditis may pass through its ordinary stages without giving rise to friction-sound at any period of the disease.   Of course, I do not mean to deny that the phenomenon may not have actually come under the cognizance of the attendant physician, from causes having no reference to its existence; such as the stage of the disease at which examination may have been made, the rapidity with which serous effusion had taken place, etc.   But the development of *frottement*, cognizable to the senses, within a period, it may be very brief, subsequent to the first stage of the disease, I hold to be a necessary pathological sequence of ordinary inflammation of the pericardium.   I further believe that in the absence of this sign, the diagnosis of pericarditis can be only presumptive; and that for the purpose of diagnosis, either physical signs are insufficient *only* when they are ill-pronounced or entirely absent at the time of examination.

That pericardial friction may be readily mistaken for other phenomena I freely admit; but this in no measure diminishes its value as a specific sign; it should rather induce a more careful study of its distinctive peculiarities, which, by such means, will be found not only cognizable, but amply sufficient to establish the identity of pericarditis where they exist.

Pericarditis may occur singly, or with intrinsic or extrinsic complications.   Thus:

1. SIMPLE PERICARDITIS ...   .   Idiopathic (?)

2. PERICARDITIS, with intrinsic complications   ...
- (a) Endo-pericarditis.
- (b) Myo-pericarditis.
- (c) Endo-myo-pericarditis.
- (d) Tuberculous deposit in the pericardium.
- (e) Cancerous ditto.
- (f) Hæmo-pericardium.
- (g) Pneumo-pericardium.

3. PERICARDITIS, with extrinsic complications, viz., with
- (a) Rheumatism.
- (b) Tuberculosis.
- (c) Pleuritis.
- (d) Pneumonia.
- (e) Pleuro-pneumonia.
- (f) Renal disease.
- (g) Hepatitis.
- (h) Scorbutus.
- (i) Gout.
- (j) Phlebitis.
- (k) Septicæmia.
- (l) Typhus fever.
- (m) Enteric fever.
- (n) Intermittent fever.
- (o) Scarlatina.
- (p) Small-Pox.
- (q) Measles.
- (r) Delirium tremens.

The history, diagnosis, and treatment of pericarditis will be most conveniently discussed under the two first mentioned heads; viz., simple pericarditis, and pericarditis with intrinsic complications. The consideration of the extrinsic complications of the disease will furnish matter for a distinct section.

Simple acute pericarditis, usually of rheumatic origin, as already shown, and most frequently met with in young subjects,* is manifested in the great majority of cases, when asso-

* Doctor Fuller declares, and in this opinion few, if any, will be found to differ from him, that rheumatic pericarditis is more common in the acute than in the chronic form of rheumatism ; in its early than in its late stages ; in youth than in advanced age ; in females than in males ; in persons of an irritable, than in those of a phlegmatic temperament ; in the weak than in the strong ; and in the fibrous than in the synovial form of rheumatism. Non-rheumatic pericarditis is more common in old age, and in men, manifestly because of its frequent association with Bright's disease ; and, finally, he adds, rheumatic pericarditis is more severe, the mortality amounting to 1 in 5·8 cases, and seldom latent ; whereas that of the non-rheumatic form is often latent.

ciated with rheumatism, within the first few days of the articular engagement. I have, however, known it to occur as late as the twenty-seventh day from the commencement of articular pain and swelling. In several instances the invasion of pericarditis was announced by precordial distress and accelerated breathing, at least twenty-four hours before physical signs of any kind were perceptible. Drs. Stokes and Mayne, as previously stated, have recorded examples of pericarditis in which symptoms sufficiently characteristic to warrant in their judgment a positive diagnosis, preceded all physical signs by periods varying from thirty to forty-eight hours [*]

I am satisfied that, with few exceptions, the rule is as above stated; namely, that the symptoms of pericarditis take precedence of the physical signs; but, I cannot regard them as sufficiently distinctive to warrant a positive diagnosis of pericarditis, pending the development of physical evidence. Indeed, these symptoms, however suggestive of pericarditis, and in association with acute or subacute rheumatism they are in the highest degree suggestive of that affection, may, individually and collectively, be found to depend upon other causes.

The symptoms of simple acute pericarditis, in its first and second stages, are of a comparatively mild character. The grave and urgent symptoms, such as aggravated dyspnœa, failure of the pulse, oppression at the precordium, indescribable anguish and fear of impending death,[†] belong rather to those cases in which the muscular substance of the heart is involved in the inflammatory process, or the organ is impeded by a copious effusion of serum into the pericardium. There is, however, more or less decided pain in the precordium. The action of the heart is accelerated, the impulse is sharp and abrupt, and the sounds, especially the second, are clear and ringing, with the addition of a characteristic rustle, distinct from friction-sound. The radial pulse is regular,[‡] and rarely above 120 in the minute

[*] See page 326

[†] Broussais *Commentaires des Propositions de Pathologie*, 1829, tom. i., p. 397, attributes this symptom to failure of the circulation in the brain.

[‡] I cannot subscribe the opinion of Louis, that irregularity of pulse is characteristic of simple pericarditis. I believe that when associated with pericarditis, irregularity of pulse indicates extension of inflammation to the substance of the heart.

Respiration is quickened in proportion to the increased rate of the heart's action; and there is usually harsh, dry, teasing cough, not accounted for by reference to the lungs, and due, probably, to reflex irritation of the larynx and bronchial surface through the coronary plexuses.

Doctor Stokes truly remarks : " If we except the pain which so commonly attends serous inflammations, the remaining symptoms of pericarditis are to be referred less to the pericardium than to the muscular fibre ;"* and Bouillaud is of opinion that rheumatic pericarditis is often painless, or very slightly painful, when not complicated with pleurisy.†

Doctor Mayne has directed special attention to epigastric tenderness as a symptom of pericarditis. It is most acute when pressure is made upwards in the direction of the heart, and was observed in a large proportion of his cases.‡

In reference to this symptom, Dr. Stokes justly remarks that its value as an aid in the diagnosis of pericarditis is much lessened by its presence, in a form scarcely, if at all, distinguishable from the latter, in acute pleuritis of the left side, and in acute gastritis. I do not think there is anything specific in this symptom, which may be noticed not only in the connexion mentioned by Dr. Stokes, but likewise, and more frequently, in cases of hepatic congestion from any cause.

The absence of pain is more characteristic of pericarditis in the complicated than in the simple form, according to Stokes ; and when present it has been occasionally observed to differ from that of pleurisy, in not being aggravated by deep inspiration, or by change of posture. I have found this last observation true in a small per centage of cases. It may be readily explained by reference to the anatomical relations of the inflamed surfaces in both cases.

If the attack complicate acute or subacute rheumatism, its general symptoms are in a great degree masked by that disease. Thus, the expression of anguish stamped upon the face, and possibly attributed to cardiac complication, may be not less rationally

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* *Opus citat.*, p. 1.
† *Traité Clinique des Maladies du Cœur*, 1835, p. 454.
‡ *Loco citat.*

accounted for by the presence of articular pain; and, similarly, heat of skin, and accelerated pulse and respiration, by the general febrile action characteristic of rheumatic fever. The peculiar abrupt impulse of the heart is, however, in association with rheumatism, eminently suggestive of incipient cardiac engagement. In the first stage of pericarditis, or that of vascular injection and suspended secretion of the serous surface, the auscultatory signs are negative, with one exception; namely, the existence of a partially suppressed *rustle*, not amounting to *frottement*. This is so characteristic that, in several instances, guided by it mainly, I have been able positively to diagnose the proximate advent of pericarditis.

The period during which this sign may continue I have known to vary from twelve to forty-eight hours; usually it lasts about twenty-four hours before friction-phenomena are declared.

The occurrence of friction-sound, with or without tactile vibration, proclaims the advent of the second stage characterized by exudation of lymph or fibrin.

Doctor Gairdner, however, is of opinion that "the friction-sound in some, even in many cases, may be the indication only of a more partial and evanescent morbid action than any to which the name of acute inflammation could with propriety be applied."*

I have already discussed at length this phenomenon (p. 289) and shall here only repeat that it is characterized by harshness of quality, prevalence at base, sensible proximity to the ear of the auscultator, liability to intensification by precordial pressure and by full inspiration, and inconstancy as to site, rhythm, and intensity.

The phenomena with which it is especially liable to be confounded are endocardial murmur, and pleuritic friction-sound limited to the antero-inferior portion of the left side of the chest.

For the distinction between endocardial murmurs and the attrition-sounds of pericarditis, Skoda† relies, on the one hand, upon the general effects of valvular disease upon the circulation

* *Edinburgh Monthly Journal,* 1851.

† *A Treatise on Auscultation and Percussion;* translated by Markham, 1853, p. 225.

and the heart, and, on the other, upon the coexistence of percussion-dulness in pericarditis.

But surely, in the early stages of valvular disease, when alone the question of diagnosis from pericarditis can arise, the derangements of circulation and the alteration in the volume and action of the heart, upon which exclusively he would base a positive diagnosis of valvular lesion and the absence of which he would regard as conclusive against that view, are, in many instances, either entirely absent, or present only in a degree quite insufficient to warrant a positive opinion.

Again, many of those changes, having reference at least to the circulation, are notoriously liable to occur in the course of pericarditis associated with extension of inflammation to the muscular substance of the heart, or with serous effusion into the pericardium.

As to precordial dulness from liquid effusion, as a means of determining an otherwise doubtful murmur to be of pericardial origin, I regard the *absence* of dulness from that cause, where attrition-murmur exists, as the rule, and its presence as the exception.

Skoda denies that the character of the murmur itself is capable of aiding our diagnosis; an opinion in which few physicians will be found to agree with him. He adds :* "If percussion indicates the presence of effusion in the region of the heart, and if the heart be not thereby displaced, we may conclude that the effusion is in the pericardium; and it then becomes very probable that the murmur also proceeds from the internal surface of the membrane." But if there be no palpable increase in the area of precordial dulness, and that in the majority of cases of pericarditis when friction-sound is audible there is no such increase I positively aver, how is the diagnosis to be made? Are we reduced to probabilities in the determination of this important question of practical medicine? I incline to think few will answer now in the affirmative.

Doctor Hope† relies upon the rougher quality of the pericardial sound as distinguishing it from those due to valvular lesion; and so well pronounced is this difference, that, according to him, where the two kinds of murmur coexist in the same sub-

* *Opus citat.*, p. 226. † *Opus citat.*, p. 174.

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ject " the one may be heard *through* the other." In such a contingency, however distinctive may be the quality of the murmur in most instances, seeing that it is remarkably prone to variation in this respect, I would rather rely upon other characteristics for its identification; and, pre-eminently, upon its rhythm, site, and liability to change of situation and character within brief periods of time.

Hope also holds that a rough, creaking, or rasping murmur with the second sound must be due to attrition, as a valvular murmur of diastolic rhythm is never of this quality. To this doctrine there are very few exceptions, notwithstanding that Dr. Walshe holds an opposite opinion.*

Da Costa declares that it is sometimes impossible to distinguish a pericardial from an endocardial sound.† I hold, on the contrary, that it is always possible to make the distinction with confidence, by carefully attending to the characteristics of pericardial *frottement* already indicated. Although in many cases, apparently very difficult, I have found the differential diagnosis in all instances feasible, at least after a second examination.

The coexistence of tactile fremitus with attrition-murmur in pericarditis, though certainly the rule, is not without exception. Enfeeblement of the heart, from any cause, by lessening its contractile energy, may entirely suspend tactile vibration, whilst only modifying, or rendering less loud and harsh, the sound of attrition. Again, fremitus, though present, may not be communicated to the hand, owing to impairment of the vibratile properties of the chest-wall, as, for example, by superficial œdema; and the intervention of emphysematous lung-substance between the pericardium and the wall of the chest, may mask a pericardial *frottement* capable of producing sensible thoracic vibration.

In many cases of valvular murmur exclusively, fremitus is also present. This is notably true of the presystolic murmur of mitral constriction. The distinction, therefore, between pericardial friction-sounds and valvular murmurs, based upon the pre-

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* *A Practical Treatise on the Diseases of the Heart and Great Vessels*, third edition, 1862, p. 118. In the fourth edition of his work just issued (1873), Dr. Walshe, however, states that aortic regurgitant murmur, the type of diastolic murmurs, is " rarely rough. '

† *Medical Diagnosis*, third edition, 1870, p. 357

sence or absence of *frémissement,* possesses only a limited value; and, independently of other considerations, excluding certain forms of valvular disease, *frémissement* affords, at least, but very equivocal evidence of pericarditis.[*]

The duration of friction-sound may vary from a few hours to three weeks. The period of its continuance is usually very brief where the cause of its suspension is serous effusion into the pericardium. In such cases it will be noticed to cease at the lower part of the precordium in the first instance, and thence to be gradually extinguished in the direction upwards, *pari passu* with the establishment of percussion-dulness.

I cannot concur in opinion with Dr. Gee, to the effect that in cases of serous effusion into the pericardium, consecutive to pericarditis, the liquid accumulation takes place in the first instance at the base of the heart and around the roots of the great vessels.[†]

In a few instances friction-sound continues to be heard at the extreme upper portion of the precordium, throughout the stage of serous effusion, and whilst the remaining portion yields femoral dulness, and the heart's sounds are everywhere masked. These are cases, however, in which the effusion has fallen short of distension of the pericardium; and the liquid gravitating to the lower portion of the sac, its superior anterior portion is allowed to come into contact with the roots of the pulmonary artery and

[*] Doctor Stokes, however, insists upon a perceptible difference in quality between the *frémissement* of pericarditis and that of valvular lesion ; adding, that the fremitus of valvular murmur is, like the murmur itself, limited to the areas of the respective orifices, whilst that of pericarditis is more diffused, and indifferent to fixed areas. This is a very just distinction so far as it applies ; but circumscribed and persistent pericardial *frémissement may* be limited to the area of one of the orifices, though I admit such is not usually the case. Again, *frémissement* arising from aneurism connected with the intra-pericardial portion of the aorta, may exhibit no relationship to valvular areas, although of limited extent. Doctor King Chambers (*Lectures chiefly Clinical,* fifth edition, 1865), lays down the following rule for the distinction of pericardial friction-sound from valvular murmur. If the ear be withdrawn from the stethoscope while still held in contact with the surface in the site of the sound under investigation, so that the lobe shall barely touch the instrument, the normal cardiac sounds will be heard, *free from murmur* in the case of pericardial friction, but *associated with murmur* if valvular disease be the cause of the phenomenon. I have put this rule to the test, and have found it practically useful.

[†] *Auscultation and Percussion,* 1870, p. 247.

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aorta. If the shoulders be depressed whilst the patient lies upon the back, the situation of friction-phenomena will, in such cases, be reversed.

In a few examples, to be noticed farther on, I have known the friction-sound of pericarditis to continue after the restoration of the patient to apparently perfect health. The rheumatic attack was of a mild character, and no symptoms were referable to the heart, nevertheless a distinct friction-sound, circumscribed within an area of very small extent, was audible at the base of the heart, continued for several weeks, and was unaltered in rhythm or intensity at the time of the patients' discharge.

Tactile fremitus is liable to considerable variation in degree of intensity, and seldom amounts to the *frémissement cataire* of Laennec. It is due to vibration of the framework of the chest, consequent upon, and therefore isochronous with, the movements of the heart. It indicates a considerable degree of roughness of the opposed serous surfaces, and hardness of the effused lymph. Dr. Stokes is of opinion that a dry condition of the surface has the effect of intensifying fremitus, if it does not constitute an essential condition of its presence. This phenomenon is never coeval with friction-sound, commencing later, and terminating earlier than the latter.

Adhesion of the parietal pericardium to the surface of the heart may follow immediately the dry stage, without the intervention of serous effusion. When it is in process of establishment, the friction-sound is irregular in rhythm, intermittent, and lowered in intensity; it next becomes single and systolic in rhythm; ceases to be heard in one or more situations within the precordium, usually persisting longest at the base of the heart, but occasionally at the apex, and is finally extinguished. The process of adhesion, where serum in notable quantity has not been effused, may commence and be completed within a period of twenty-four hours

The *positive* diagnosis of cohesion between the parietal and visceral pericardium, from evidence afforded by that condition itself, independently of personal observation of the previous illness, or a reliable history of its progress, is not possible in the present state of the science of physical diagnosis.

Palpitation, supposed to indicate irritative reaction under restraint, was long regarded as a symptom of adhesion of the pericardium to the heart. Morgagni,[*] however, after carefully analysing a great number of reported cases of palpitation alleged to arise from this cause, and also several cases of adherent pericardium noted by Valsalva and himself, arrives at the conclusion that palpitation cannot be fairly attributed to adhesion of the pericardium *alone* in the adduced cases; in all of which, other, and frequently many, causes combined to produce it. He observes, moreover, that, with a single exception, in all the cases witnessed by himself in which this was the only tangible morbid condition discovered, palpitation was *not* present. The exception was that of a patient who had suffered from palpitation, and in whom, after death, adhesion of the pericardium to the roots of the great vessels was found. To this latter circumstance, as tending to obstruct the outflow of blood by constricting the vessels, rather than to the general adhesion of the pericardium, he inclines to attribute the palpitation which had been observed during life.

Senac[†] also doubts that adhesion of the pericardium has any *special* influence in causing palpitation, which he attributes in a general way to various forms of external and internal irritation of the heart, of which adherent and thickened pericardium may be one; especially if, at the same time, it make pressure on the roots of the great arteries.

Corvisart[‡] regards, as symptoms of adherent pericardium, frequent flushings, and a sensation of dragging in the region of the heart, due to downward traction of the pericardium and heart by the diaphragm during inspiration, and upward traction of the diaphragm by the heart in systole. He mentions three kinds of adhesion; namely, 1, by exuded lymph; 2, by direct union without a uniting medium, which he regards as the usual form in gout and rheumatism; and 3, long filamentous bands.

Skoda, however, holds,[§] but not very strongly, that the diag-

[*] *The Seats and Causes of Disease*, translated by Alexander, book ii., letter xxiii.
[†] *De la Structure du Cœur*, l. iv., ch. xi.
[‡] *A Treatise on the Diseases and Organic Lesions of the Heart and Great Vessels*, translated by Hebb, p. 32.
[§] *Opus citat.*, p. 327, note.

nosis is practicable; he bases it mainly upon suppression of apex-pulsation, and an alleged upward and inward displacement, with fixation, of the apex of the heart. He mentions as a further sign of adhesion, systolic retraction of the intercostal space at a point corresponding to the apex, and to a slight extent of one or two spaces next above; but this implies adhesion of the pericardium to the costal pleura.

I cannot regard these signs, taken absolutely, as of much value. Displacement of the apex upwards and inwards may be the result of partial retraction of the left lung, already adherent to the cone of the pleura and to the external surface of the pericardium; whilst suppression of the apex-beat may be due to fatty disease of the heart, or enfeeblement of the organ from other causes, such as typhus, or septic blood-poisoning. But if the other causes of upward displacement of the heart be excluded, and especially if pericarditis be known to have preceded, a fixed position of the apex-point at or near the nipple may be regarded as eminently suggestive of adhesion.

Laennec alludes* to the sign mentioned by Sanders; namely, retraction or pitting of the epigastrium below the left costal cartilages, followed by elevation with pulsation of the same point. He declares, however, that although he had repeatedly looked for it where adhesion of the pericardium actually existed, he had never observed it.

This is likewise my own experience. I have repeatedly observed pericarditis pass through its entire course, from the pre-friction or dry stage, to final suppression of friction-sound, and I have not even once witnessed the sign mentioned by Sanders. I have frequently observed epigastric pulsation of such a character as might be readily mistaken for the phenomenon in question; but, I feel bound to add that, I have observed this most frequently in hypertrophy with dilatation, where no adhesion of the pericardium existed.

Laennec does not consider the diagnosis feasible. Such is likewise the opinion of Bertin and Bouillaud.

Hope, while asserting that the diagnosis of adherent pericardium cannot be made with positive certainty, considered that

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* *Mediate Auscultation*, Forbes' translation, p. 666.

strong probability of its presence may be assumed, if, with hypertrophy of the heart, there be abnormal prominence of the precordium, without downward displacement of the organ ; and, at the same time, a jogging or tumbling movement of a double character, synchronous with the sounds of the heart. This latter he regards as being, in a diagnostic sense, the more valuable of the two signs, and as warranting a very strong presumption of cohesion of the pericardial surfaces.*

I cannot attach the importance to this sign which Dr. Hope has done, as evidence of adhesion of the pericardium. I have repeatedly found it present where no adhesion existed, as proved by examination of the body after death; and I have rarely missed it where considerable hypertrophy with dilatation of the left ventricle had taken place (case of John Hogan). In the latter case, unless the hypertrophy be associated with fatty metamorphosis, there is invariably a second or *diastolic* impulse present (case of William Hogan). This I consider due to the energetic character of ventricular expansion at the commencement of diastole, combined with the extensive range of its forward movement; and I incline to regard it as identical with the "jogging movement" of Hope.

Doctor Stokes has likewise failed to identify this peculiar movement of the heart, in connexion with adhesion of that organ to the parietal pericardium.

The diagnosis is not attended with equal difficulty when, at the same time, adhesion of the pericardium to the chest-wall and to the diaphragm exists. Under these circumstances systolic dimpling of the cutaneous surface at the point of apex-pulsation, and, generally, to the extent of endo- and exo-pericardial adhesion, also coinciding, possibly, with pitting at the epigastrium and retraction of the lower end of the sternum, may be observed.

According to Friedreich,† diastolic collapse of the jugular veins will be found in many cases to alternate with these phenomena ; owing, as he surmises, to elongation of the superior cava, and consequent suction of the jugulars during the descent of the

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* *Opus citat.*, p. 194.

† Niemeyer's *Text-Book of Practical Medicine*, 1869, vol. i., p. 891.

diaphragm after systole. If adhesion of the pericardium to the adjacent portions of the anterior wall of the chest exist, the edges of the lungs will be precluded from advancing, as in the normal state, during inspiration; and the area of superficial precordial dulness will be found, therefore, to have undergone no diminution at the acme of full inspiration.

Laennec mentions* dulling of the second sound as evidence of adhesion of the anterior surface of the heart to the pericardium. But this is certainly an error; and it seems an inference from his theory of the causation of the second sound, namely, contraction of the auricles, rather than the result of clinical observation.

I have frequently observed resolution of the first sound to follow pericarditis, when the previous course of the disease forbade all doubt as to the actual existence of adhesion of the pericardium (case of M. Nolan); and I incline to account for this anomaly by reference to the impediment which extensive adhesion must present to the movement of systolic ascent of the apex. But, whatever value the explanation may possess, I am satisfied as to the fact, that recent adhesion of the pericardium to the heart is attended with resolution or reduplication of the first sound, which, in such cases, is likewise somewhat less clear in tone.

Walshe mentions† upward extension of the area of precordial dulness, even to the lower edge of the second costal cartilage, consecutive to pericarditis, as a sign of adhesion.

I have not met with an example of this kind either during or consecutive to pericarditis, where indubitable evidence of liquid effusion into the pericardium did not also exist. I can, however, with Dr. Walshe, conceive displacement of the heart upwards, but in a minor degree, by copious effusion; and its being permanently fixed above its normal level by subsequent adhesion to the pericardium.

I consider, then, that whilst simple cohesion of the parietal and visceral surfaces of the pericardium cannot be diagnosed positively, a very strong presumption of the existence of this

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* *Mediate Auscultation*, Forbes' edition, p. 665.
† *Opus citat.*, p. 47.

condition would be warranted, were the apex of the heart permanently displaced upwards, the first sound resolved and the impulse-element dulled, and a clear history of antecedent pericarditis obtained. If, with or without such history, systolic pitting of an intercostal space at the point of apex-pulsation, and of the epigastrium, with absence of variation in the relative extent of precordial dulness and vocal vibration during full inspiration existed, I should have no hesitation in concluding that both pericardial and pleuro-pericardial adhesion had taken place. Exaggerated systolic depression of the epigastrium, and of the lower end of the sternum, would indicate the twofold condition of general agglutination of the pericardium, and hypertrophy of the heart.

The effect of adhesion of the pericardium upon the heart, and its influence upon the duration of life, were regarded by the older writers as of the most unfavourable character. This was, no doubt, due to their failure to recognise, or at least to appreciate in its full significance, the less striking lesion of the valves ; which, coeval with adhesion of the pericardium to the heart, and constituting with it the compound result of long antecedent endo-pericarditis, was either entirely passed over, or received only cursory notice in the examination of the heart after death. The more obvious lesion of the pericardium was promptly recognised, and, as affording an apparently simple and ready explanation of the increased volume of the heart, was associated, in the crude pathology of that time, with the latter change, as cause with effect.

Morgagni held that cohesion of the opposed surfaces of the pericardium was a cause of dropsical effusion.* Baillie was of opinion that close and general adhesion gave rise to oppression and difficult breathing ; whilst Corvisart thought that it must be ultimately fatal by derangement of the function of the heart.†

Bouillaud considered hypertrophy of the heart and œdema to be necessary consequences of simple adhesion of the pericardium, whilst atrophy was a necessary result of compression of the

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* *De Sedibus et Causis Morborum.*
† *On Diseases of the Heart,* translated by Hebb.

heart by thick layers of false membrane.* Cheevers† and Barlow‡ declare that atrophy of the heart is the usual and normal result of obliteration of the pericardial sac by adhesion; the former adding, that when dyspnœa and dropsy occur in connexion with it, they are due to consecutive contraction of the arterial orifices, and venous engorgement. Barlow admits that engorgement of the right side of the heart, of the veins, and general dropsy, may be the consequences of adhesion of the pericardium; but he traces the connexion between them through impairment of respiration, which he attributes to restricted movement of the ribs and diaphragm.

Complete obliteration of the sac of the pericardium by adhesion, leads inevitably to hypertrophy with dilatation of the heart, according to Hope.§ He regards hypertrophy as the result of increased nutrition and muscular development of the heart, consequent upon the increased efforts in the performance of its function, imposed upon that organ by adhesion of its surfaces. The difficulty under which the heart is now required to "function," consisting in the impediment to free movement entailed upon it by adhesion of the pericardium to its entire surface, leads, as in the case of all muscles similarly circumstanced, to hypertrophy of its substance. Dilatation, on the other hand, is due to a state of persistent engorgement of the cavities of the heart, and excentric yielding of its walls, partly the result of antecedent inflammation and softening of its substance, but in a still greater degree, of the centrifugal traction to which it is subjected by universal adhesion of its surface, combined with a process of gradual shortening in the elements of the connecting medium. Limited adhesion, engaging only a small portion of the surface of the heart, may, in the opinion of Dr. Stokes, be the cause of localized dilatation of one of the cavities of the heart, through the centrifugal traction exercised by it.‖

Beau upholds the same view. Dr. Stokes observes,¶ "With-

* *Traité Clinique des Maladies du Cœur,* observation iv.
† *Guy's Hospital Reports,* vol. ix.
‡ *Medical Gazette,* 1847.
§ *Opus citat.,* p. 192.
‖ *Opus citat.,* p. 13.
¶ *Opus citat.,* pp. 11 and 12.

out denying that a general adhesion may induce hypertrophy and dilatation, experience leads me to doubt that such an effect necessarily, or even commonly, follows the condition indicated. I have often found the heart in a perfectly natural condition, with the exception of an obliterated pericardium." " It has been stated to me by Professor Smith, that he has found general adhesion of the pericardium coinciding with atrophy, or with hypertrophy of the heart, in a nearly equal frequency. In some of the cases of atrophy, the change was simple, consisting essentially in a diminished volume, with perhaps a paler colour of the heart, while in others a true fatty degeneration had commenced. In another series, the heart showed the fatty degeneration invading, more or less completely, the entire of the cardiac walls. And it is a remarkable fact, recorded by the same observer, that he has always found ossification of the pericardium, which we may hold as the extreme of the obliterating process, attended with atrophy of the heart."

Doctors Barlow,[*] Cheevers,[†] and Gairdner,[‡] have arrived at conclusions on this subject, not materially differing from that expressed in the foregoing extract, and tending to show that the doctrine, previously stated, as propounded by Hope, is by much too general.

Doctor Gairdner justly criticizes Hope's conclusions, which he shows to have been drawn from erroneous premises; for, in the five cases adduced by Hope as examples of hypertrophy of the heart *consequent upon* adhesion of the pericardium, there were other and less questionable causes of that morbid change in operation; viz., valvular disease in various forms.

It is but right, however, that whilst agreeing in opinion with Dr. Gairdner as to the insufficiency of the evidence upon which Hope's doctrine rests, and regarding that doctrine as essentially erroneous, I should add, that the cases adduced by Dr. Gairdner,[§] in justification of his dissent, are to me by no means conclusive. Of his first series of ten cases, three must be omitted

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* *Medical Gazette*, 1847.
† *Guy's Hospital Reports*, vol. ix.
‡ *Edinburgh Monthly Journal of Medical Science*, February, 1851.
§ *Loco citat.*

for want of reference to the actual state of the heart ; whilst the remainder, which are examples of adhesion with atrophy, possess not much value, because they are all examples of wasting chronic disease, viz., cancer, phthisis, diabetes mellitus, granular kidney, and cirrhosis of the liver.

In series 2, consisting of two cases, illustrative of adhesion of the pericardium with cardiac hypertrophy, the latter condition was accounted for by the coexistence of valvular disease.

Series 3 (three cases) is composed of examples of adhesion with hypertrophy, and unaccompanied by valvular lesion. Of these, No. 1 is not admissible, owing to an imperfect report of essential details. No. 2 is an example of Bright's disease ("urine albuminous, specific gravity 1·009") with hypertrophy of the heart, and partial adhesion of the pericardium ; and in No. 3, in which there were hypertrophy and dilatation, with partial adhesion, a satisfactory examination of the patient had not been made before death, nor was the state of the kidneys mentioned in the report of the autopsy.

Of Dr. Gairdner's thirteen cases of uncomplicated adhesion of the pericardium, there were at least nine in which "not the slightest hypertrophy was present, and in two or three the heart was remarkably small." He concludes that free movements of the heart within the pericardium, are necessary only in emergencies demanding more vigorous action of the central organ of the circulation, such as severe bodily exertion, or mental emotion ; and he would have it inferred that it is only when such causes are superadded to adhesion, that hypertrophy of the heart need be apprehended. But, surely, the causes above mentioned, are of themselves capable, independently of adhesion, of giving rise to hypertrophy. Irrespectively, therefore, of the exception which may be taken to the doctrine promulgated, the conclusion would seem unwarranted by the premises. Nor can I acquiesce in the opinion expressed by the same eminent writer, to the effect, that partial adhesion at the apex may be as effective in producing hypertrophy of the heart, as complete and general adhesion.

With the view of contributing in some degree towards the settlement of the much debated question, as to the effect of ad-

hesion of the pericardium upon the nutrition and volume of the heart, I have collected the following statistics, bearing on the subject, from sources whence information of a reliable character, or least open to suspicion, may be reasonably sought. These statistics I have reduced to tabular form, under separate heads, indicating: 1, the rheumatic or non-rheumatic origin of the pericarditis ; 2, whether it was accompanied or not by valvular disease; 3, by liquid effusion ; and, 4, followed or not by hypertrophy.

The tables are three in number. The first contains, in epitome, the examples of pericarditis published in the Reports of the London Pathological Society ; the second, those given in the Reports of the Pathological Society of Dublin ; and the third contains a summary of the cases which have come under my personal notice.

PERICARDIUM: TABLE I.

London Pathological Society.

Cases.	Rheumatic.	Non-Rheumatic.	Valvo-Disease.	Hydro-peri-cardium.	Hypertrophy.	Observations.
(1) Prescott Hewett, vol. i. Dec. 6, 1847	—	1	—	—	Hypertrophy and dilatation of right ventricle; left ventricle small and not thickened.	General adhesion of pericardium.
(2) Dr. Williams, vol. i. April 17, 1848	—	1	—	1	Left ventricle and right ventricle dilated	Partial adhesion of pericardium.
(3) Dr. Parkes, vol. ii. Feb. 19, 1849	—	1	—	—	—	Wound of pericardium, from œsophagus, by swallowing a sword; pneumo-pericardium; general exudation of lymph.
(4) Dr. Bence Jones, vol. iv. Dec. 7, 1852	—	1	—	—	Of left ventricle	Originating in two circumscribed abscesses between liver and diaphragm, which found entrance into pericardium, extensive adhesion.
(5) Dr. R. R. Robinson, vol. iv, Dec. 7, 1852	—	1	Thickening of aortic, & atheroma of the aorta.	1	1	A man, aged forty-three; intemperate; fall from ladder. *Post mortem*; congenital opening in pericardium, through which left auricular appendix protruded; fatty heart; calcareous coronary arteries; cirrhosis of liver. Bright's disease; partial adhesion of pericardium by bands of false membrane.

						Remarks
(6) Dr. Bristowe, vol. v. March 21, 1854	—	—	—	1	—	Double pleuropneumonia; exudation of thick layer of lymph; palor and fatty degeneration of outer layer of ventricles, which was in contact with false membrane.
(7) Dr. Habershon, vol. vi. Dec. 5, 1854	1	—	Constrict. of mitral orifice, cohesion of valve, and small nodular growths upon it	—	And dilatation of left ventricle; dilatation of right ventricle	General adhesion of pericardium; hydatids encroaching upon right auricle and ventricle.
(8) Dr. Cholmeley, vol. vi May 15, 1855	—	1	—	—	1	Pericardium almost universally adherent; heart enlarged and fatty; large aneurismal pouch at apex of left ventricle, walls of which were composed of endo- and exocardium only; angina pectoris and calcified coronary arteries; fatty degeneration of outer and inner layers of muscle.
(9) Dr. Quain, vol. vii. Nov. 6, 1855	—	1	—	—	1	Pericardium partially adherent and calcified; calcareous deposit in aorta and in mitral valve.
(10) Dr. G. Hewitt, vol. viii. Nov. 18, 1856	1	—	Mitral valve warty and incompetent	1	General, especially of right ventricle	Much effused lymph, but no adhesion.
(11) Dr. Hawksley, vol. viii. May 19, 1857	—	1	—	3 oz. of pus	—	Remittent fever, and eruption on skin; thick and vascular layer of lymph on visceral pericardium only.
(12) Dr. Obré, for Dr. Boyd, vol. ix. Dec. 15, 1858	—	—	—	—	—	A lunatic, aged sixty-three; no reliable history; adhesion of pericardium, and small abscess at apex.

PERICARDITIS : TABLE I.—*Continued.*

Cases.	Rheumatic	Non-Rheumatic.	Valve-Disease.	Hydro-peri-cardium.	Hypertrophy.	Observations.
(13) Dr. Markham, vol. ix. April 6, 1858	1	—	Valves generally slightly thickened, and competent	—	—	Graves' disease, aggravated; enlarged thymus, pressing on pulmonary artery and causing systolic murmur; patch of fibrinous transformation in wall of left ventricle, and a similar change, in a greater degree, of left papillary muscles.
(14) Dr. John Ogle, vol. ix. April 6, 1858	—	1	—	—	1	A large cyst beneath visceral pericardium on posterior surface of right ventricle, containing altered blood and laminated clot; judged to be of long standing, and due to rupture of branch of calcareous coronary artery; general adhesion of pericardium with calcareous deposits in false membrane; texture of heart soft.
(15) Dr. Wilks, vol. xi. April 7, 1860	—	1		1	—	A boy of fourteen; had scarlatina six months before; had peritonitis and ascites at period of death; pericardium distended with liquid, nearly filled left pleural cavity, displacing lung backwards and inwards, and concealing it entirely, save apex, and contained a pint of clear serum; parietal pericardium was much thickened by deposit of several layers of false membrane, the inner of which was soft; heart similarly covered, the outer layer being soft and the subjacent layers firm; the deepest tightly embracing the heart, and adhering to the visceral pericardium, which was thickened, and sending fibres into substance of heart; heart small for size and age of patient, and

Reference						Remarks
						appeared as if it had been strangled by the firm coating of lymph upon it ; valves and endocardium, generally, quite healthy.
(16) Dr. Bristowe, vol. xi. Dec. 4, 1860	—	1	—	—	1	A boy of thirteen ; tubercular pericarditis, ascites, and pleuritic effusion ; tubercular deposit in anterior mediastinum ; universal adhesion of pericardium ; the adhesions thick and fibriod, and in them much tubercular matter, in form of cheesy masses and laminae of great extent ; walls of left ventricle thin, and cavity dilated ; right ventricle very thick and dilated ; auricles dilated.
(17) Dr. Gibb, for Dr. David, vol. xiii. Oct. 15, 1861	1	—	General endocarditis	1	1	A coloured man, aged forty-two ; died in St. Patrick's Hospital, Montreal ; acute rheumatism of ten days' duration, with pleuritis and pericarditis ; pericardium contained eight ozs. of turbid serum, and both its surfaces were covered with thick dense flakes of fibrin "like tripe" ; there was no adhesion. Intense endocarditis on both sides ; heart weighed forty-six and a-half ounces ; it was fifteen and a-quarter inches in circumference, and six inches in length.
(18) Dr. Peacock, vol. xiv. April 7, 1863	—	1	No mention	—	No mention	A man, aged thirty-one ; carcinoma of lung, intestine, kidneys, and supra-renal capsules ; pericarditis with nodules of carcinoma in false membrane. No notice of size of heart, or state of valves, and no information given as to whether there was adhesion of pericardium, or effusion into its cavity.
18	4	12	5	5	11	18

PERICARDITIS : TABLE II.

Dublin Pathological Society.

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-peri-cardium.	Hypertrophy.	OBSERVATIONS.
(1) Dr. Law, vol. i. April 3, 1841.	1	—	Aortic and mitral	—	1	Patient aged twenty-two ; had repeated attacks of rheumatism ; heart weighed thirty-six ounces ; complete adhesion of pericardium.
(2) Mr. O'Ferrall, vol. i. April 2, 1842.	1	—	No mention	—	1	A boy of eleven ; had scarlatina six months, and rheumatic fever, two months before death ; adhesion of pericardium by bands of lymph.
(3) Dr. Corrigan, vol. i. Dec. 3, 1842	—	1	Mitral (?)	1	—	A boy of eighteen ; pericard. so much expanded by serous effusion as to reach first rib, and coated internally with rough papillated lymph ; slight thickening of mitral valve ; no adhesion.
(4) Dr. Corrigan, vol. i. Jan. 13. 1844	—	1	No mention	1	No mention	A railway labourer ; pericarditis of four months' duration ; three pints of dark-coloured serum in pericard. and some adhesion at base ; heart rough by effused lymph ; "pine-apple heart."
(5) Dr. Lees, vol. i. Jan. 11, 1845	—	1	No mention	1	No mention	A woman of sixty-eight ; left circumscribed pleuritis with pericarditis ; hæmorrhagic effusion into pericard. which was reticulated ; no adhesion.
(6) Dr. Corrigan, vol. ii. Dec. 7, 1850	—	1	No mention	1	No mention	A girl of seventeen ; overworked ; died of dysentery ; pericarditis diagnosed by *frottement*

Reference						Remarks
(7) Dr. Banks, vol. ii. Jan. i 1848	—	1	—	1	1	audible when she lay on her back, but inaudible when she sat up and leaned forward; a few flakes of unorganized lymph were found, and a few patches, organized, on the outersurface of the right ventricle. Eight ounces of liquid in pericardial sac. The history showed that the lymph had remained unorganized for a period of six weeks; no adhesion. A man of sixty; pleuro-pneumonia; heart enlarged and fatty; slight atheroma of aorta; calcareous plate, two inches diameter, in false membrane on right ventricle; nearly complete adhesion of pericardium.
(8) Dr. M'Dowel, vol. iii. April 16, 1853	1	—	Mitral thick and incompetent	Six ounces (of blood)	1	A boy of twelve; had acute rheumatism three years before; chorea, with mitral reflux and hypertrophy, a year later; at end of another year dyspnœa, pericardial *frottement*, and friction-sound. Heart covered, except at apex, by several layers of false membrane, in some places three-quarters of an inch thick; adhesion by bands of false membrane at base.
(9) Dr. Mayne, vol. iii. March 31, 1855	1	—	Mitral slightly incompetent	—	1	A boy of thirteen; acute rheumatic endocarditis at eleven; a year and nine months later another similar attack, and five months subsequently died in syncope; universal adhesion of pericardium.
(10) Dr. Mayne, vol. iii. Same date	—	1	No mention	Filled with pus	No mention	An infant of five months; pericarditis after measles; pericardium very vascular, and filled with pus; no adhesion.

PERICARDITIS : TABLE II.—*Continued.*

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-peri-cardium.	Hypertrophy.	Observations.
(11) Dr. M'Dowel, vol. iii. Dec. 5, 1856	1	—	Slight thickening of aortic and mitral	1	—	A child of eleven years; death on the ninth day; a few ounces of serum in pericardium; surfaces vascular; some false membrane; no adhesion.
(12) Dr. Henry Kennedy, vol. iii. Jan. 8, 1859	No mention	No mention	—	—	1	There was hypertrophy, with dilatation, and fatty disease of substance of heart; nearly complete adhesion of pericardium.
(13) Dr. Hudson, vol. iii. April 20, 1861	No mention	No mention	No mention	1	No mention	Pericarditis, with inflammation; softening of the heart; friction-sound ceased three or four days before death. *Post mortem*: serum with coating of lymph; softening of heart, and infiltration of its substance; "grumous fluid." Dr. Hudson remarks that the signs of extension of inflammation to the heart were, increased precordial pain and anxiety, tendency to syncope, failure of radial pulse, irregularity of heart's impulse, and cessation of attrition-sound without increase of precordial dulness.
(14) Dr. Banks, vol. iii. Dec. 6, 1862	No mention	No mention	Aortic and mitral inadequacy from dilatation	—	1	A man, aged eighteen years; hypertrophy to thirty-eight ounces with dilatation; universal adhesion of pericardium.

Dr. Henry Kennedy, April 16, 1864 (15)	No mention	No mention	No mention	—	Acute pericarditis, without the usual accompaniment of *frottement*, although pericardium was covered with reticulated false membrane. Dr. Kennedy inquires whether this may not have been due to rapid action of heart. No adhesion.
Dr. Gordon, vol. iii. March 21, 1868 (16)	1	—	1	—	Secondary pericarditis, with exo-pericarditis; exo-pericardium adherent to chest-wall; hence says Dr. Gordon, the great dilatation with hypertrophy; whereas, in ordinary adhesion with compression of the heart, atrophy is the rule; adhesion.
Dr. Lyons, vol. iii. March 30, 1867 (17)	—	No mention	1	1	Acute pericarditis in the fourth week of typhus, and fatal on the twenty-seventh day; serum and lymph were found in pericardium; no adhesion. (?)
Dr. Hayden, vol. iii. May 2, 1868 (18)	—	—	1	—	A man, aged twenty-two; hepatic disease and general dropsy; cartilaginous and calcareous transformation of pericardium; atrophy and dilatation of heart; general adhesion.
Dr. Jennings, vol. iv. part. i. Jan. 9, 1869 (19)	No mention	No mention	No mention	1 (?)	A man, aged forty-two; sudden death; tuberculosis of lungs and pleural effusion; external adhesion of pericardium; left ventricle fatty; aorta highly atheromatous; complete adhesion of pericardium by means of false membrane half an inch thick.

PERICARDITIS : TABLE II.—*Continued.*

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-peri-cardium.	Hypertrophy.	Observations.
(20) Dr. Eames, vol. iv. part i. Jan. 16, 1869	1	—	—	1	1	A child at nurse, neglected ; pericardium extended to right nipple, and upwards to second rib on left side ; it had pushed up lung above level of clavicle ; it contained twelve ounces of amber-coloured serum, and was covered with false membrane ; purulent deposits in various parts ; no adhesion.
(21) Dr. T. E. Little, vol. iv. part i. Jan. 16, 1869	—	1	—	1	—	A man, aged thirty-one, with curvature of spine, an inmate of the Sligo Lunatic Asylum ; signs of pericarditis had continued three months ; pericardium twenty-four inches in circumference ; vascular externally, thickened, and coated internally with lymph, and containing sixty-five ounces of dark-coloured serum ; mediastinal glands large and tubercular ; left pleural adhesion ; no pericardial adhesion.
(22) Dr. Stokes, vol. iv. part i. Jan. 30, 1869	1	—	Double aortic	—	1	A man twenty-five years of age ; long subject to yearly attacks of rheumatic arthritis ; a ploughman by occupation ; double murmur at aortic orifice ; throbbing of carotids, and loud pericardial *frottement* ; both aortic murmurs disappeared before death ; whereas, according to Dr. Stokes, this is rarely the case with regard to the aortic diastolic murmur ; pericardium was found greatly thickened and covered with

23	9	9	8	10	11	28
Dr. MacSwiney, vol. iv. part i. Jan. 29, 1870 (25)	1	—	—	Recent deposit on aortic and mitral	No mention	lymph in great quantity, disposed evenly, and in rugged masses; one segment of the aortic valve was calcified and incompetent; general hypertrophy; heart, with pericardium, weighed four pounds and two ounces; the left ventricle was one and a half inch thick; partial adhesion of pericardium. Dr. Stokes is of opinion that the great and general hypertrophy in this case was due to the laborious occupation of the poor man, and which he himself thought had agreed with him. A man who had had rheumatism four years previously, was attacked with pleuro-pneumonia and pericarditis; pericardium was found thickened, and both its surfaces coated with rough lymph; foetid gas escaped on opening the pericardium, which, having been distended, was supposed to contain liquid; partial adhesion of pericardium.

PERICARDITIS : TABLE III.

The Author's Cases.

Cases.	Rheumatic.	Non-Rheumatic.	Valve-Disease.	Hydro-pericardium.	Hypertrophy.	OBSERVATIONS.
(1) J. Doyle	1	—	—	1	—	Pericarditis twelve days after rheumatic invasion ; no head symptoms ; recovery.
(2) J. Doran	1	—	Regurgitant, mitral, double aortic	—	1	Rheumatism sixteen years ago, and pericarditis *with* return of articular pains ; no head symptoms ; recovery.
(3) L. O'Brien	1	—	Mitral obstruction and reflux	1	1	Pericarditis seventeen days after articular pains ; no head symptoms ; recovery.
(4) Pat Macdonnell	—	1	Double mitral, and aortic, and tricuspid	1	1	Had rheumatism nine years before, and again several times, but not recently ; no head symptoms.
(5) Pat Costello	—	1	Mitral obstruction	—	1	Long subject to rheumatic gout, but pericarditis consecutive to organic disease ; no head symptoms ; death.
(6) Margaret Bligh	—	1	—	1	1	Palpitation one year before, after retrocession of cutaneous eruption ; no head symptoms ; recovery.

	8	5	7	10	9	Remarks
(7) Mary Nolan	1	—	Temporary mitral inadequacy	1	—	Pericarditis two days after articular pains; murmur had entirely ceased when discharged; slight nocturnal delirium.
(8) John Smith	—	1	—	1	1	Pulmonary emphysema, and hypertrophy of right ventricle; head symptoms due to this; death; no *post mortem*.
(9) Anne Lalor	1	⋅|	Mitral reflux	1	1	Had had rheumatism; hypertrophy judged by heaving impulse; no *post mortem*; murmur developed in last illness; no head symptoms.
(10) Kate Kavanagh	1	—	—	1	—	Recent case.
(11) James Walsh	1	—	Double aortic and single mitral	1	1	Pericarditis, not preceded by articular swelling, four days after exposure; valve-disease of long standing, as judged from hypertrophy of heart.
(12) Pat. Moore	1	—	—	⋮	—	Recent case.
(18) James Farrell	—	1	—	1	1	Chronic phthisis, pericarditis and pleuritis; no adhesion.
13	8	5	7	10	9	13

Of the preceding cases, in number 54,
    Valvular disease did not exist in 23.

Of this latter number hypertrophy was present in 14 :
    General adhesion   8
    Partial adhesion   2
    Calcification of false membrane   4

Hypertrophy did not exist in 7.

Atrophy existed in 2 :
    Strangulation by thick layers of false membrane   1
    Cartilaginous and calcareous transformation of false membrane ...   1

Of the cases in which hypertrophy did not exist, there was no adhesion in   5
  ,,   ,,   ,,   ,,   adhesion in   2
  ,,   ,,   ,,   ,,   recovery in ...   2

Of the total of cases, in number 54 :
    Hypertrophy was present, associated with adhesion of the pericardium,
        and unconnected with any other assignable cause of its existence,
        such as chronic valvular or renal disease, or arterial atheroma, in   7

Valvular disease existed in 20 :
    Thickening of valves generally   2
    Mitral, aortic, and tricuspid obstruction and reflux   1
    Aortic obstruction   1
    Aortic obstruction and reflux   1
    Mitral obstruction   2
    Mitral reflux   6
    Mitral obstruction and reflux   1
    Mitral reflux, and aortic obstruction and reflux   6
    No mention is made of the condition of the valves in   9
    There was temporary mitral regurgitation in   1
    There is doubt as to the condition of the valves in   1
    Pericarditis was associated with rheumatism in   21
        ,,   not associated with rheumatism in   26
    Hydro-pericardium existed in   25
    Hypertrophy in   31

Hypertrophy did not exist in 9 :
    No adhesion of the pericardium   5
    Adhesion of the pericardium   2
    Recent cases ; recovered   2

From the preceding data, it appears that out of a total of fifty-four cases of pericarditis, collected from the three sources indicated, only seven can be adduced in support of the doctrine that simple adhesion of the pericardium is competent to give rise to hypertrophy of the heart; but, even these are not re-

ported in such a manner as to inspire complete confidence. In several of them the urine was not tested, nor was disease of the kidneys excluded by necroscopic examination. I therefore feel warranted in concluding, as I had been for a long time led to infer from personal observation, that, contrary to the teaching of Hope, when hypertrophy of the heart is met with in association with adherent pericardium, the connexion is, in the majority of cases, purely accidental. Some other less questionable cause of hypertrophy is present, such as valve-lesion, chronic renal disease, or atheroma of the aortic arch. Dr. Barclay is of the same opinion, regarding aortic atheroma and renal disease as the cause of simple hypertrophy of the heart in the majority of cases.* But, even with this deduction, examples of hypertrophy are to be met with, and a few such are included in the preceding list, in which increase in the weight and volume of the heart would seem to depend upon adhesion of the pericardium exclusively. How are such cases to be explained ? I believe that the greater number, if not the entire, of such cases, resolve themselves into examples of pericarditis occurring in infancy or childhood, in a robust constitution, and where no mechanical impediment to the growth of the heart existed. Under these circumstances, the persistent stimulus to nutrition, from inflammatory irritation of its surface in the first instance, and, subsequently, from the mechanical irritation of inflammatory products to which the heart is subjected, must issue in excessive development of its substance, provided only the conditions for a proportionate increase in the supply of pabulum, and the capacity for its appropriation by the tissue of the heart, be likewise present.†

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* *Medico-Chirurgical Transactions,* vol. xxxv.

† Dr. Moxon (Lectures on Analytical Pathology, lecture ix., *Medical Times and Gazette,* November 26th, 1870), is of opinion that "under irritation the tubular elements (nerve, muscle, capillary vessel) never multiply, but waste away, or rapidly break down." But, surely, under irritation physiological activity is quickened, and exaggerated structural development is thereby induced.

Dr. Todd (*Clinical Lectures by Beale,* 1861, lecture xvi. p. 482) holds that hypertrophy and dilatation of the heart are usual pathological sequences of adhesion of the pericardium. I cannot agree in this opinion. For the discussion of the subject generally, the reader is referred to the chapter now under consideration.

If the coronary arteries be free from obstruction, and the heart be not constricted by false membrane of a thickness and power of resistance sufficient to prevent its growth, then simple pericarditis, occurring within the first or second period of life, when nutrition is active, growth rapidly progressive, and buoyancy of spirits inductive of vigorous movements of the body, will inevitably lead to hypertrophy of the heart. I believe that these arguments apply, with scarcely less force, to the period of adolescence, provided only the constitution be vigorous.

If, on the other hand, even in the early periods of the life of healthy subjects, pericarditis give rise to constriction of the nutrient vessels of the heart, of its substance, or of both, arrest of growth, or atrophy, will be the result, irrespectively of the character of the constricting medium.

Bouillaud is of opinion* that adhesion, as such, does not interfere with the play or function of the heart.

Doctor R. W. Smith,† holds that calcification of the pericardium is more often associated with atrophy than with hypertrophy of the heart. He published the case of a man aged thirty-nine years, in whom the ventricles were encircled by an osseous belt, one inch broad, imbedded in their substance, the auricles were hypertrophied, but the ventricles had undergone no particular change.‡ In a case of ossified pericardium, Louis found the volume of the heart a little reduced.§

Abercrombie gives the following particulars of a case of pericarditis with adhesion and ossification of the false membrane. A man, aged forty-two, after exposure to cold and fatigue, exhibited symptoms indicative of acute intrathoracic inflammation, from which he recovered. He subsequently suffered from dyspnœa, palpitation, and general dropsy, and four years after the date of first illness he died. The autopsy revealed extensive adhesion of the pericardium to the surrounding structures, also general adhesion of the pericardium to the heart, and, in the uniting medium, osseous plates; " the heart

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* *Traité Clinique des Maladies du Cœur*, 1835, p. 454.

† As quoted by Dr. Stokes.

‡ *Dublin Journal of Medical Science*, vol. ix. p. 411, July 1st, 1836.

§ *Memoires ou Recherches Anatomico-Pathologiques*, 1826, p. 298.

was not at all enlarged, or in other respects diseased." He adds the opinion that dilatation supervenes on inflammation of the heart, and is usually accompanied by adhesion of the pericardium.*

Doctor W. Budd declares† he has seen many cases of adhesion, often general, without organic alteration of the heart, or impairment of its function.

Bouillaud expresses an opinion to the effect that a heart may be reduced to half of its volume, or even less, by the compression of thick layers of false membrane.‡ Walshe is of opinion that atrophy of the heart may result from thick layers of false membrane firmly embracing the heart, and pressing on the coronary arteries so as to interfere with the circulation through them.§ Finally, Dr. Barclay declares that adhesions of the pericardium are more frequently associated with dilatation than with hypertrophy.‖

From the preceding evidence, the conclusion seems inevitable, that the doctrine of hypertrophy from simple adhesion can no longer be maintained.

Abercrombie, discussing the effects upon the heart of adhesion of the pericardium, says, " In the cases which have gone on for a considerable time, the heart is generally enlarged ; this enlargement seems to be, in many cases, confined to the left ventricle, and to consist of an enlargement of the cavity, without thickening of the parietes." Yet the case of enlargement which he gives, presented valve lesion also, as shown by dissection. He adds, " but enlargement may exist without adhesion, and adhesion without enlargement,"¶ and suggests that inflammatory engagement of the membrane only, or of the membrane and the muscular substance conjointly, may explain the difference. I believe that when the muscular substance of the heart has been seriously implicated in the inflammatory process, whether pri-

* *Transactions of Medico-Chirurgical Society of Edinburgh,* vol. i., 1821.

† *Library of Medicine,* vol. v., p. 195.

‡ *Opus citat.*

§ *Opus citat.*

‖ *Medico-Chirurgical Transactions,* vol. xxxv.

¶ *Loco citat.*

mary or secondary, degeneration and atrophy of the organ, at a more or less remote period, are the necessary consequence.

Wilks declares* "that simple pericardial adhesions produce no visible ill consequences;" adding, "the loose cellular adhesions have no appreciable influence on the action of the heart, but that the thickened pericardium of cartilaginous consistency, investing the heart closely, arising from an inflammation at an early period of childhood, does lead to obstruction of the circulation, and then to dropsy, after the ordinary manner of heart-disease." He holds, also, that concurrent myocarditis may be the cause of subsequent dilatation of the heart by softening, the adhesions may involve the great vessels within the pericardium, and cause constriction of these, and its usual sequelæ; and thickening, he alleges, may extend into the cellular tissue of the anterior mediastinum and neck, and press on the veins, thereby causing cyanosis of the face, neck, and upper extremities.

The following cases (given in abstract) are published by Wilks in confirmation of the foregoing opinions:

Case 1. A man, aged twenty-one, ailing and short-breathed from childhood, subject to epistaxis and swelling of the feet, with lividity of the surface. No *bruit*. On dissection, universal adhesion of the pericardium, with bony deposit on the right side was found; enlargement of the auricles, but thinning and contraction of the ventricles, muscular tissue of the heart pale and flabby; valves healthy.

Case 2. A boy, aged nine, had scarlatina three years previously. Now livid, with dyspnœa and general dropsy. No *bruit*. P. M. General adhesion of the pericardium, which was as thick as leather from deposit of successive layers of false membrane, of which the internal were softer; the left ventricle was small and thin; the right chambers dense by infiltration of fibrinous tissue; valves healthy.

Case 3. A boy of twelve; some months before was attacked with dyspnœa after exposure; general dropsy gradually set in, accompanied by lividity; there was enlargement of the liver, but no cardiac *bruit*. P. M. General adhesion of pericardium, strongest and thickest on right side; heart normal as to size,

* *Guy's Hospital Reports*, third series, vol. xiv., March, 1871.

valves healthy; liver of " nutmeg " character; lungs adherent to chest and to pericardium.

Case 4. A girl of sixteen, had rheumatism in childhood, since when symptoms of heart-affection had manifested themselves; died suddenly of pulmonary congestion and pneumonia. P. M. Lungs universally adherent to chest and to pericardium; latter attached to heart by false membrane, most firmly to right ventricle; all the chambers of the heart were thin except the left auricle; muscular substance pale and flabby; valves healthy and competent; mitral slightly thickened but competent.

Case 5. A man, aged thirty-six, had been in hospital a year previously for an affection of the heart, which yielded no abnormal sign except obscurity of the sounds; re-admitted with extreme dyspnœa and palpitation; there was slight dropsical effusion, but no *bruit*. P. M. Pericardium universally adherent, one-fourth of an inch thick, and indurated; heart generally enlarged; valves healthy; liver " nutmeg "; lungs generally attached to chest and pericardium.

Case 6. A man, aged twenty-four, had rheumatism some years previously, since when he suffered from dyspnœa, cough, and expectoration; after recent exposure dyspnœa became extreme, and was accompanied by lividity; no cardiac *bruit*. P. M. Lungs congested and universally adherent to chest and pericardium, which was firmly attached by false membrane to heart, and thickened; left ventricle normal; right ventricle hypertrophied; valves healthy, except aortic, which presented a few vegetations, but were competent.

The cases above narrated certainly afford no support to the opinion, that simple adhesion of the pericardium is competent to produce hypertrophy of the heart; nor do they in a greater degree favour the doctrine that atrophy of the heart is the normal consequence of this pathological change. Indeed, the conclusion to which they lead is rather of a negative kind, namely, that cohesion of the opposed surfaces of the pericardium has no positive influence as such upon the volume of the heart, and that when, in association with it, hypertrophy or atrophy of that organ is met with, other, and it may be, latent causes, are in operation to explain the connexion. Amongst the causes of

hypertrophy, valvular disease holds the foremost place; but, of this cause, dilatation of the chamber *behind* the affected valve in the course of the circulation, is always a concomitant result.

Chronic renal disease holds the next place in the order of requency; but, owing partly to the occasional absence of prominent symptoms having reference to the kidneys, and, in some measure, likewise, to inattention to the state of these organs, the association of renal disease with cardiac hypertrophy has been frequently lost sight of. Disease or tissue-degeneration of the coats of the arteries, especially the ascending portion of the aortic arch, by imposing additional propulsion-duty upon the left ventricle, may be, and frequently is, a cause of hypertrophy of that chamber Such hypertrophy, however, will be found to be simple, or unassociated with dilatation, unless softening or tissue-degeneration of the heart be a concomitant lesion.

I believe, as already stated, that pericarditis occurring in early life when the growth of the heart is in active progress, and involving only surface-irritation of the organ, whether arising directly from the inflammatory process, or, mediately, through the accumulated products of inflammation, and interfering in no degree with the coronary circulation, leads directly to general hypertrophy of the heart. I further hold that it will, of necessity, be followed by hypertrophy, provided only the patient survive for a period of, at least, one to two years, and that no countervailing agency come into operation, such as phthisis, or marasmus from any other cause.

Under the opposite conditions, namely, constriction of the heart and coronary arteries by exuded and organized fibrin, to such a degree that the circulation and tissue-nutrition of the organ is seriously impaired, the growth of the heart will be arrested in the child, and early death from failure of the circulation will be the necesssary consequence. In the adult the same result will follow, but at a more remote period from the date of invasion, and as a consequence of actual and progressive atrophy of the heart.

Between the two conditions above sketched, there is conceivably one of an intermediate grade, in which the inflammation is neither sufficiently active to stimulate the heart to abnormal

excitement, nor its products in sufficient quantity to cause mechanical irritation or disturbance of circulation. In such cases, of which examples will be presented in the sequel, the heart will be found to have undergone no alteration of volume whatever.

If inflammation have, at the same time, extended to the substance of the heart, and caused softening of the organ, dilatation will be the result, with or without hypertrophy, according to the extent to which the nutrition of the heart, or its nutrient supply, has been compromised by the preceding inflammation. I regard external adhesion of the pericardium, whether to the lungs or to the sternum, as of much less consequence than tissue-softening of the heart, in determining dilatation of its chambers. The cooperation of both these causes, as can be readily conceived, will give rise to maximum dilatation, usually with hypertrophy, general or partial, because of the concurrent stimulus to nutrition. Where, with such a combination of organic lesions, produced in early youth and associated with valvular disease, life has been long protracted, an example of the *cor bovinum* or ox-heart is likely to be the result. A morbid specimen of this character, and exemplifying in the highest degree the alterations of volume and capacity in the heart which these causes are capable of producing, was exhibited to the Pathological Society of Dublin, by Dr. Stokes, in January, 1869.*

Exuded lymph, the product of acute inflammation, if not already organized, is liable to absorption on the pericardial as on all other serous surfaces; but the membrane remains permanently opaque by interstitial cell-proliferation. It must be admitted, however, that organization and persistency, with or without adhesion, of lymph effused on the surface of the pericardium as the result of acute inflammation, is the almost universal rule; and, further, that of the cases in which lymph so produced is organized, ninety per cent., at least, furnish examples of mutual adhesion of the serous surfaces.

Mayne was of opinion that complete resolution may take place in the second stage of pericarditis, irrespectively of the extent of surface engaged.†

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* See Table II., Case 22, p. 358.

† *Dublin Journal of Medical and Chemical Science*, vol. vii., 1835.

Such was likewise the opinion of Kirkes.*

Fuller holds that pericarditis of limited extent and mild character, may undergo complete resolution in the second stage, leaving only "white spots" as the traces of its former existence ;† whilst Gairdner denies the resolution of acute pericarditis without adhesion.‡

The exuded lymph is of a pale rather than a blanched hue ; it usually presents the appearance, at first, of disseminated, semi-liquid, minute droplets, which, after a period of twelve to twenty-four hours, become solid, and usually, likewise, dotted with sanguineous *puncta*. In very acute cases, the exuded fibrin is from the first of a blood-red tint. The opposed surfaces are, in most cases, coextensively, but not equally, affected, and the exudation may be universally diffused, or limited to one or more distinct and separate portions of the pericardial surfaces. In the latter case, the surface of the right auricle, the anterior surface of the right ventricle, and the right and left apex of the heart, to a variable extent, are the parts most usually affected.

Bouillaud has described, under the designation of " pine-apple heart," a condition of that organ, in which the changes consequent on pericarditis are presented in an exaggerated form. The heart is hypertrophied, and its entire surface is covered with minute conical masses of solid and organized lymph, resembling thus a pine-apple in appearance, no adhesion between the visceral and parietal pericardium having taken place.

In limited or circumscribed adhesion of the pericardium, the union of the surfaces is usually effected by bands of lymph, sometimes an inch or more in length, extending between them, and, but very rarely, by direct agglutination ; whereas, universal adhesion is effected almost exclusively by close and direct union of the surfaces. In the former case it would seem as if the points of attachment were not sufficiently numerous to restrain the free movements of the heart, by which, consequently, the plastic lymph is drawn out in the form of filaments or bands,

* *Medical Gazette*, 1849
† *Diseases of the Chest*, 1862.
‡ *Monthly Journal of Medical Science*, February, 1851

corresponding in length to the entire, or to a certain proportion, of the range of relative displacement of the surfaces, according to the degree of strength or capacity for resistance which they respectively possess.

It is scarcely conceivable that adhesion of this character can exercise any palpable influence upon the volume of the heart. I have never found, in connexion with it, either hypertrophy or atrophy of the organ, which was not susceptible of other and more satisfactory explanation.

General adhesion is effected by the mutual apposition and fixation of the two serous surfaces, through the medium of plastic lymph, which passes through the successive stages of vascularization, solidification, and contraction. The effused lymph at first opposes no obstacle to the free movement of the two surfaces, and soon acquires sufficient consistence to yield a friction-sound. When first heard, this sound is faint, and not specially harsh ; it soon, however, acquires this quality, and in a ratio as to time and intensity proportionate to the increase of consistence of the lymph. As adhesion proceeds, owing to the increasing impediment to the free movements of the heart, the sound of friction becomes less and less representative of the rhythm of these movements ; whilst, at the same time, it assumes a still more harsh and grating character, in consequence of the increasing density of the exudation ; ultimately, a single sound of systolic rhythm, and usually basic in site, corresponding to the more powerful movement of the heart, is alone audible. Adhesion may be now regarded as all but complete ; and at this period the uniting medium will be found condensed, contracted, and vascular.

In a few exceptional cases *frottement* has continued audible for several consecutive months over a very limited area, whilst in all other situations it had long previously ceased (case of the girl Young). Presumably, in these cases, adhesion had been completed at all points, save that at which friction-sound was still audible ; but why the process of adhesion should have failed in this situation, and, as may be inferred from the existence of distinct friction-sound, without the intervention of liquid or gas by which the surfaces might have been kept apart, I am unable

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to say. It occasionally happens that lymph of a pasty and aplastic character, is found smeared over portions of the heart's surface to which it loosely adheres. In such cases, pericarditis usually complicates general adynamic disease of some kind, such as typhoid pneumonia, typhus or typhoid fever, the low forms of scarlatina and variola, puerperal fever, phlebitis, delirium tremens, or pyæmia; and adhesion entirely fails. Detached flocks of pale fibrin are likewise, in such cases, not uncommonly found floating in the turbid serous contents of the pericardium.

I have not met with an example of typhus complicated with pericarditis, and Dr. Stokes has witnessed only one such case.* When pericarditis occurs in connexion with any of the diseases characterized by the typhoid condition, I should expect that it would be *latent* as to symptoms; but even in such cases the physical signs are singularly constant.

On this subject Andral remarks:† " In the several cases which we have reported, pericarditis was announced by an assemblage of symptoms which rendered its diagnosis sufficiently easy.

" We are now about to adduce cases in which the most prominent of these symptoms, pain, did not exist; and it was only in some degree, by proceeding by exclusion, that one could arrive at a recognition of inflammation of the pericardium."

The cases alluded to were examples of latent pericarditis; and the remarks bearing on them, just quoted, show clearly that Andral, at the date (1829) of issue of the second edition of his great work, from which the extract is made, was unacquainted with the pathognomonic sign of pericarditis.

Fuller truly remarks‡ that rheumatic pericarditis is seldom latent, whilst that of the non-rheumatic kind is often so.

Sir T. Watson gives the particulars of a very interesting case of latent pericarditis, illustrative of the features, negative and positive, of that form of the disease.§

According to Dr. Stokes, pericarditis, when latent, is invariably

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* *Opus citat.*, p. 80.
† *Clin. Méd.*, vol. i., p. 23.
‡ *Diseases of the Chest*, p. 513, *et sequent.*
§ *London Medical Gazette*, vol. xvi., part i., April 11th, 1835.

of the dry form, and that the more urgent symptoms by which the disease is usually characterized, are due, not to the inflammation of the serous membrane, but to one of its consequences, namely, serous effusion into the sac.

In the majority of cases of acute pericarditis, the plastic material constituting false membranes, connecting bands, or loose shreds found in the pericardium, would be correctly designated as a *deposit* from a liquid exudation identical with the liquor sanguinis.

Rindfleish, however, states that the product of acute inflammation of serous membranes is twofold; a fibrinous deposit from the liquor sanguinis, as already described, which adapts itself to the various irregularities of the surface, forming a cast of it, without being organically united with it, and ultimately disappearing by fatty degeneration, liquefaction, and absorption, and very rarely undergoing caseation and no further change. Beneath this the epithelium is tumefied, its nuclei proliferate and burst from their respective particles, the *débris* of which is floated away in the general liquid contents of the cavity, to undergo ultimate solution and absorption; whilst the nuclei, greatly multiplied, enter into the formation of the subjacent plastic and organizable stratum. Of this latter element, however, the great bulk consists of embryonic connective tissue, derived from the proliferating connective tissue corpuscles of the subserous structure. This becomes organized, blood-vessels are formed in it by linear apposition and tunneling of cells, and by entrance of blood from neighbouring blood-vessels. Finally, the opposed surfaces come into contact, and are united by continuity of structure, and extension of blood-vessels from the one to the other through the connecting media; the embryonic tissue becomes fibrous, and contracts, vascularity is diminished, absorption of all unorganized material is completed, and solid permanent union is the result.*

It occasionally, however, but rarely, happens, that plastic lymph alone is effused upon the serous surfaces, constituting " dry" pericarditis. It likewise occasionally happens that upon the serous surfaces fibrin is primarily effused, from which, in

* *Pathological Histology,* Sydenham Society's edition, vol. i., p. 305.

process of organization, serum is subsequently expressed, and trickling to the most dependent portion of the sac, constitutes a veritable but limited serous effusion. It may be a question whether, in the majority of instances, this is not the mode in which primary serous collections of limited amount, and coeval with initial *frottement*, are formed.

In ordinary cases, therefore, *frottement* indicates not only the presence of lymph, of which it is directly symptomatic, but likewise that of its usual concomitant, serum. In the early stage of the affection, the latter is present in comparatively small quantity in most instances, and insufficient to prevent mutual contact of the serous surfaces at one or more points of their extent; it gravitates to the dependent portion of the sac, according to the posture of the body. Hence, when the patient lies upon the back *frottement* may be heard over the entire superficial precordium, and in the sitting posture only in the upper part, to an extent inversely proportionate to the height of the liquid level within. If the patient lie upon the abdomen, friction-sound may be heard, extensively in the left infra-scapular region, save in those cases, comparatively numerous, in which the posterior portion of the pericardium happens to be unaffected by the inflammatory process.

Doctor Latham maintains, but I cannot agree with him, that the exocardial murmur is "neither abolished, nor abated, nor otherwise altered in its character by the serum effused within its cavity."[*] In the absolute sense in which this statement is intended to be applied, it is certainly incorrect; but that serum in small quantity is usually present in all cases save those of "dry" pericarditis, is no less certain. Hence, in great measure, the modification of *frottement* to which change of posture is known to give rise.

In cases involving difficulty of diagnosis, as between pericardial friction-sound and endocardial murmur, Sir D. Corrigan assigns great value to the evidence, positive or negative, afforded by the patient's assuming the prone or stooping posture[†] If the sound cease to be heard when the patient sits up, and leans for-

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[*] *Lectures*, vol. i., p. 139.
[†] *Reports of Dublin Pathological Society*, vol. ii., December 7th, 1850.

ward, he would regard it as exocardial in origin, and due to pericarditis; and if still audible in that posture, he would regard it as endocardial.

This rule, though undoubtedly useful in the process *par exclusion*, and capable of yielding valuable aid in a certain per centage of difficult cases, is of only limited application, and involves at least two fallacies. Thus, in dry pericarditis, the pseudomurmur may be even intensified by assumption of the stooping posture; and by the effort involved in assuming that posture, it occasionally, though rarely happens, that an endocardial murmur is masked or even suspended. This is especially true as applied to functional mitral murmur. (See pp. 285 and 287.)

Latham holds that cessation of *frottement* in pericarditis, un-associated with the signs of liquid effusion, implies, in all cases, without exception, cohesion of the surfaces of the pericardium. Yet, with marked inconsistency he maintains that cessation of an endocardial murmur would indicate resolution or absorption of the exuded fibrin.

I cannot admit either proposition absolutely. Pericardial *frottement* may be finally suspended without adhesion, when it has been originally due to the presence of aplastic fibrin, in connexion with any of the various forms of general adynamic disease. In such cases, whilst adhesion fails from want of organizing energy in the tissues and the recent exudation, *frottement* is not uncommonly suspended or abolished, owing to typhoid softening and impaired contractile energy of the heart. In the second place, inasmuch as endocardial murmur, associated or not with pericarditis, may be, and frequently is, due to other causes than endocarditis, as already stated, for example, temporary debility, and local yielding of the walls of the left ventricle during systole, the cessation of it cannot be accepted as proof of the absorption of the products of endocarditis.

Serous effusion may increase, and usually does so, to the extent of completely separating the surfaces of the pericardium, and suspending friction-sound and *frémissement*. When this has occurred, the third stage, so called, of pericarditis, or that of liquid effusion, has been established; but this stage implies only the increase of an effusion which had previously existed. On

the other hand, instead of increasing, it may undergo decrease, and the surfaces, being now in extensive and uninterrupted contact, will become united through the medium of organized lymph previously deposited upon, or exuded from, the serous surfaces. If the lymph effused be of a low type, or aplastic in character, it may remain unorganized, and either feebly attached to the surface of the pericardium, or as loose shreds within its cavity, adhesion having in no degree taken place.

Such aplastic exudations are of usual occurrence in the pericarditis which complicates chronic renal disease and adynamic fevers of a septic character. They are in process of slow fatty disintegration. In chronic pericarditis generally, which is usually of non-rheumatic origin, there is, as justly maintained by Laennec, rarely a false membrane ; when such is present, it is soft, friable, and like a layer of thick pus, and accompanied by a turbid puriform liquid effusion. This form of the disease is, he alleges, always general, engaging the entire surface of the pericardium ; the exuded lymph is likewise redder than in the acute form, the redness being due to many minute blood-points. When pericarditis arises from, or is associated with, diffuse inflammation, then effusion into the pericardium is likely to be purulent.*

In connexion with acute pericarditis, blood is occasionally found, even in considerable quantity, in the sac of the pericardium. An example of this kind is mentioned by Dr. Latham.†

Andral furnishes a still more striking example. A man, aged thirty-one years, was attacked with severe radiating and burning pain in the region of the heart, followed by numbness extending down the front of the left arm and forearm, and alternating with excruciating pain in the same parts, during which respiration became greatly oppressed, the heart's action tumultuous and irregular, pulse failing, and surface cold. After a few minutes, pain ceased and numbness returned ; the pulse was now regular but small ; breathing less embarrassed, but the action of the heart still quick and energetic, and heard over the entire front of the chest.

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* See a case by Dr. Law : *Reports of Pathological Society of Dublin*, vol. i., p. 42.
† *London Medical Gazette*, vol. iii., p. 7, December 6th, 1828.

On the fifth day of illness the patient was convalescent; but, on the following day great weakness and symptoms of sinking suddenly appeared; the heart's impulse was no longer perceptible; the precordium was extensively dull, and the patient died in the course of the night. About two pounds of blood were found in the pericardium, and shreds of false membrane covering its surface.*

In such cases the diathesis is hæmorrhagic, and a state of hypinosis, or blood-attenuation, exists; evidence of which may be found in the tumid state of the gums, and in the liability of the patient to bleeding from the mouth, nose, bladder, and intestinal canal, and to ecchymosis of the conjunctivæ after slight irritation.

Bouillaud truly remarks that simple blood-staining of the false membrane of pericarditis is not uncommon, and that it is most frequently the immediate result of imbibition from a blood-stained liquid effusion.

Gaseous exhalation into the pericardium, the direct result of inflammation of its serous lining, has been likewise observed.

Graves details the following example of pericardial pneumatosis. A girl, aged twenty-five, was suddenly attacked with severe and general pain in the abdomen, resembling peritonitis; five days subsequently a tumor of a conical shape, yielding distinct fluctuation, and dull on percussion, appeared at the epigastrium, extending downwards within two inches of the umbilicus, and into either hypochondrium; two days later purging suddenly set in; the epigastric tumor, now slightly reduced in size, no longer yielded fluctuation, and was tympanitic on percussion; purging continued; the patient became gradually weaker, and much wasted; the tumor had entirely subsided after an interval of twenty days from the date of its first appearance, when she was suddenly attacked with violent pain in the region of the heart, accompanied by palpitation, and a sensation of burning beneath the ensiform cartilage. The precordium was now found to be tympanitic on percussion; double *frottement* was heard over the base of the heart, and occasionally a metallic click inside the nipple; next day, emphysematous crackling

* *Clinique Méd.*, second edition, 1829, vol, i., observ. iii., p. 15.

was heard, and after a further interval of two days, a regular metallic tinkling, synchronous with the heart's action, took the place of the latter phenomenon, and superseded all other physical signs, except a slight *bruit de soufflet* at the mamma. Death took place one month from the date of first illness. A large abscess was found in the left lobe of the liver, communicating with the stomach by three several openings, and with the pericardium by another through the diaphragm. The pericardium was inflamed, thickened, coated with lymph, and contained about two ounces of pus.*

The pericardium in this case became inflated from the stomach, whilst the pus found in it proceeded from the hepatic abscess, and was the immediate cause of pericarditis.

Doctor Stokes gives a case of primary pneumatosis of the pericardium, so interesting that it deserves to be reproduced in its entirety. In his own words, " A young man, of lymphatic temperament, had laboured under an attack of acute pericarditis for a few days before I saw him. On my first examination, he presented the usual signs of dry pericarditis, with a considerable effusion of lymph of the ordinary consistence. The rubbing sounds, though loud and distinct, had nothing unusual in their character, and the patient suffered but little distress. After two or three days I saw him again, and found that his state had become very much altered. His appearance was haggard and worn, and he complained of extreme exhaustion, which he attributed to a total deprivation of sleep. This was induced by the extraordinary loudness and singular character of the sounds proceeding from the cardiac region; for though up to this period the rubbing sounds were distinctly perceptible by means of the stethoscope, the patient was quite unconscious of their existence. They had suddenly, however, become so loud and singular, that the patient and his wife, who occupied the same apartment, were unable to obtain a moment's repose. On examination, a series of sounds was observable which I had never before met with. It is difficult or impossible to convey in words any idea of the extraordinary phenomena then presented. They were not the rasping sounds of indurated lymph, or the

* *Clinical Medicine,* vol. ii., p. 233.

leather creak of Collin, nor those proceeding from pericarditis with valvular murmur, but a mixture of the various attrition murmurs with a large crepitating and a gurgling sound, while to all these phenomena was added a distinct metallic character. In the whole of my experience I never met so extraordinary a combination of sounds. The stomach was not distended by air, and the lung and pleura were unaffected, but the region of the heart gave a *tympanitic bruit de pot fêlé* on percussion, and I could form no conclusion but that the pericardium contained air, in addition to an effusion of serum and coagulable lymph."*

I have not met with an example of this singular condition, but judging from those above given, I conclude that it would be distinguished by the following peculiarities. 1st. Precordial tympany without displacement of the heart, and distinguishable from that of the stomach, by the *absence*, higher up, of normal precordial dulness. Pulmonary emphysema, would, however, modify the last mentioned diagnostic, but this may be readily distinguished by its proper signs, especially the *downward* displacement of the heart, a change of position the reverse of that which the heart would assume from the pressure of an inflated stomach. 2nd. Pericardial *frottement* of extraordinary intensity, and audible to the patient and to bystanders. 3rd, but of less diagnostic significance than the former, an overwhelming sensation of oppression and sinking,

Whatever the nature of the pericardial effusion be, whether serum, pus, blood, or gas, it may prove rapidly fatal by impeding the action of the heart, either by its absolute quantity, or by still further embarrassing an already feeble organ. It may, on the other hand, undergo absorption, and its liquid portions be quickly and entirely removed, leaving, in the case of purulent or sanguineous effusion, a solid deposit upon the serous surfaces. This deposit is paste-like, pale yellow in colour, and of a granular and non-coherent texture where the antecedent effusion has been purulent; but, where blood has been extravasated, the residuum, after absorption of its liquid portion, consists of fibrin and the *débris* of the blood-corpuscles, tinted in various shades of red, according to the time elapsed since the primary effusion.

* *The Diseases of the Heart and the Aorta*, 1845, p. 21.

In recent cases the deposit presents the decided tint of blood; and, after a protracted interval, during which it has been subjected to a process of gradual absorption, the colouring matter may be entirely removed, and the new surface may be found of a colour only a shade deeper than that of a primary exudation of fibrin. Extravasation of blood into the pericardium, of non-traumatic origin, is usually consecutive to inflammation, and of much later date; indeed, its most frequent source is the vascular plexus developed in the recently organized lymph upon the serous surfaces. Hence, the appearance resembling the granulated surface of a healing ulcer, to which, when examined at an early period, it has been aptly compared; or where the subjacent exudation of lymph has assumed the character of conical growths, and become solid in texture, the surface presents the appearance of the dorsum of a neat's tongue smeared with blood. The term *hæmorrhagic* is used to designate this form of pericarditis, which is usually the consequence of an *accident* of ordinary serous inflammation, occurring in a diathesis favourable to sanguineous effusion. Rokitansky declares that it is of most frequent occurrence in connexion with tuberculous pericarditis. Examples of sanguineous and purulent collections in the sac of the pericardium will be found recorded in Tables I. and II. (See pp. 350 and 354.)

The title *dry pericarditis* is intended to designate a form of the disease in which the morbid process is arrested in the second stage, and liquid effusion does not take place. It is usually, at the same time, slow as to progress, and latent as to symptoms.

Doctor Stokes declares that he has rarely met with it where pericarditis was not complicated by " inflammation in various organs and different tissues."* In most of the cases which have come under my notice there was preexisting renal disease, with hypertrophy of the heart, and the friction was characterized by extreme loudness, extensive diffusion, and long continuance. Indeed, I cannot recall an example of this form of pericarditis which proved fatal, in which the friction-sound did not continue up to a brief period preceding death, when failure of the heart was the cause of its cessation.

* *The Diseases of the Heart and the Aorta*, p. 68.

Doctor Stokes has noticed its continuance in one case for a period of sixteen days. Examples of no less duration will be found amongst the cases appended to this chapter.

I have further noticed that in all these cases the primary disease, of which pericarditis was a complication, had already given rise to dropsical effusion elsewhere, usually of the feet and legs, previously to the occurrence of pericarditis. Has this effusion exercised a *derivative* influence upon the pericardium in process of inflammation ? Judging from analogy, I am disposed to answer in the affirmative. The chronic character of the disease, and the absence of liquid effusion in these cases, may likewise serve to explain the ill pronounced character of the symptoms, or latency of the affection. The intensity of the *frottement* may be accounted for in part by the total absence of liquid from the sac, but chiefly by the coexistence of hypertrophy of the heart. The physical signs distinctive of dry pericarditis are persistent clearness on percussion beyond the normal area of precordial dulness, distinct *frémissement*, and loud, long-continued, and extensively diffused friction-sound. Judged by its associations, it is of most unfavourable augury.

In a few examples already adverted to, I have met with dry pericarditis under different circumstances, and with a very different result. The patients were affected with rheumatism of a mild character, and exhibited no cardiac symptoms whatever. Nevertheless, circumscribed basic *frottement* existed, continued unaltered through convalescence, and, at the end of several weeks, when the patients were discharged " cured," was still present and no less distinctly pronounced. I regard this as due to permanent roughening of a limited extent of the pericardial surfaces by lymph deposited in a granular form, and arrested in its process of organization at the stage in which its vascular supply sufficed barely for the maintenance of its vitality, and was incapable of establishing adhesion by extending into contiguous tissue. So far as I know, friction-sound of this kind is permanent, and the condition which causes it leads to no alteration in the volume or structure of the heart, and in no degree shortens life.

Hydro-pericardium, or collection of serum in the pericardial

sac, would mean, according to the definition of Corvisart, all such accumulations exceeding in quantity six ounces.   He mentions a case in which eight pounds of serum were found in the pericardium.   The quantity, however, rarely exceeds two pints. After death a few drachms of serum are always to be found in the pericardium, even when death has surprised the party in a state of perfect health.   This is the result of *post mortem* condensation of the halitus present in the pericardium, as in all serous cavities, during life; the source of which is the epithelial surface, and the purpose to be served, that of facilitating the gliding movements of the opposed surfaces upon one another in the process of "functioning" of the invested viscera.   The accumulation of a larger quantity is morbid, and the result of derangement of the balance between secretion and absorption.   It is not difficult to conceive how such derangement may arise in connexion with inflammation, associated as that process is with vascular engorgement, and deranged vascular pressure and tension.   If, at the same time, owing to attendant pyrexia, impaired secretory function of other organs normally engaged in the elimination of water from the body be a concomitant symptom, as is usually the case, excessive secretion of serum into the pericardium, and absolute, as well as relative, retardation of absorption, must be the result.

The diagnosis of liquid effusion in the pericardium, when in small or even moderate quantity, is usually attended with considerable difficulty.

Galen* held that in such cases the palpitation of the heart communicates a sensation as if the organ were moving in a fluid He also mentioned, as symptomatic of hydro-pericardium, great languor, drooping of the eyes and somnolence, palor and lividity of the lips and finger-ends, great loss of flesh, weak, small and intermitting pulse, and dyspnœa, with extension of the neck.

Morgagni mentioned only two symptoms as possessing diagnostic value; namely, a sense of weight on the heart, and of squeezing oppression at the precordium on making exertion. In the case of the virgin recorded by him,† the colour of the face was good, sleep undisturbed, bowels and menstruation regular,

* Quoted by Morgagni.   † *Epistola* xvi

breathing equally easy in the standing, supine, and in either lateral posture, pulse natural, and no palpitation, pain in chest, or cough. He denied that swooning and cough are usual.

Senac* speaks of dropsy of the pericardium as a disease " that is frequent, difficult to be known, and more difficult to be cured."

Corvisart regarded, as the symptoms of hydro-pericardium, lividity of countenance, anxiety and sense of weight, and tumultuous pulsation at the precordium, great dyspnœa in the horizontal posture, syncope, palpitation, small, feeble, frequent, and occasionally irregular pulse, great debility, extended dulness, often protuberance of the left side, œdema of lower limbs, and occasionally puffing of the front and left side of the chest.

He once felt, but never saw, localized fluctuation in the fourth and fifth intercostal spaces, as mentioned by Senac. He attached some diagnostic value to threatened suffocation from lying on the right side, and, still more, to the sign which he claimed to have discovered; namely, pulsation of the heart at various and distant points, owing, as he believed, to the floating of the organ in a liquid medium, and within an enlarged pericardium, as determined by the movements and posture of the body.

Doctor Latham says that where effusion into the pericardium coexists with exocardial murmur, which, according to him, is usually in no degree modified by it,† an undulatory movement in the third and fourth left interchondral spaces may be seen, and vibration felt in the same situation. He adds that vibration is more frequent, and often unaccompanied by undulation, but undulation never without vibration.

All these alleged symptoms of effusion into the pericardium are eminently equivocal, with the exception of precordial dulness,

* Lib. iv., ch. v.

† Andral (*Anatomie Pathologique*, p. 520, foot note) mentions a case in which two and a-half pints of liquid were found in the pericardium, yet friction-sound continued up to the time of death. In this case, however, there was considerable hypertrophy of the heart, and the usual posture of the patient was that of stooping, the elbows resting on the knees. It is possible, therefore, as nothing is stated to the contrary, that partial adhesion may have taken place between the heart and anterior wall of the pericardium. Again, it is not at all improbable that increase of the effusion took place in the death-struggle, and after the final examination of the patient's chest, as frequently happens.

and localized precordial œdema and protrusion. Even these symptoms are not conclusive, but they are much more significant than the others included in the preceding category.

The symptoms and signs of liquid effusion into the pericardium may be classified under two heads; namely, equivocal and unequivocal. Under the head of signs I propose to include only the evidence which may be elicited by auscultation and percussion.

Of the preceding list of equivocal symptoms, to which should be added dysphagia, I will discuss here only that observed by Senac, and subsequently sanctioned by the authority of Corvisart and Latham ; namely, intercostal fluctuation and dysphagia.

I have never witnessed intercostal fluctuation in connexion with hydro-pericardium, and, therefore, I have no personal knowledge of the value which should be assigned to this alleged symptom. Judging, however, from my general knowledge of allied morbid conditions, I incline to the conclusion that this symptom will be manifested only where the intercostal muscles are paralyzed and protruded ;* and as this is most likely to be the case in pyo-pericardium, it becomes a question whether intercostal fluctuation, limited to the precordium, should not be regarded as pathognomonic of purulent collections in the pericardium.

Dysphagia has been noticed by Stokes, Mayne, and others, in connexion with liquid accumulations of large amount in the pericardium.

It is conceivable on anatomical grounds, that from such a cause difficulty in the deglutition of solids may arise ; but, as a matter of fact, such difficulty has been noticed by the most competent observers. Viewed in the abstract, however, it cannot be regarded as possessing much diagnostic significance ; because, in the first place, it may be due to morbid changes within the œsophagus itself, either accompanied by, or independent of, pericardial effusion ; or to the pressure of aneurismal, cancerous, or other tumors in the posterior mediastinum ; and, secondly, because most assuredly many examples, I would say, indeed,

* See Dr. Stokes on the " Signs of Empyema," *Dublin Medical Journal*, vol. ix.

the greater number, of copious effusion into the pericardium, are unaccompanied by this symptom.

If, on the other hand, by the history of the case, these possible causes of dysphagia, intrinsic and extrinsic, be excluded, and pericardial effusion be, on other grounds, presumably present, then the coexistence of dysphagia assumes much corroborative value. When present, dysphagia is usually associated with weak and high-pitched voice; but it may occur in the first stage of pericarditis complicated with diaphragmatic pleurisy, in which case it would not, of necessity, be accompanied by dysphonia. Amongst the unequivocal symptoms of copious effusion into the pericardium, I would place precordial enlargement, determined by measurement taken from the posterior fold of the axilla to the mesial line of the sternum on each side, whilst the arm is raised above the horizontal line, and the hand rests upon the head. The existence of relative enlargement, so determined, will possess more significance if presented on the side of the body on which the arm is less freely used, because of the possible error arising from excessive muscular development. It will appear in the sequel, that if this phenomenon coincide with certain physical signs, to be mentioned presently, the diagnosis of pericardial effusion of large amount may be confidently made.

The physical signs of effusion which are of equivocal value are negative and positive :

Negative.

1. Absence of respiratory sound to an abnormal extent in the precordium.

2. Absence of cardiac sounds of normal distinctness.

3. Absence of, or very feeble cardiac impulse.

Positive.

1. Dulness on percussion limited to the precordium.

2. Displacement, temporary or permanent, of the point of apex-pulsation.

3. Friction-sound synchronous with the movements of the heart.

4. Disproportionate extension of dulness upwards.

The absence of respiratory sound over an increased precordial area, taken alone, possesses no special significance; because it may be due to tumor in the anterior mediastinum, cancer of the left lung, circumscribed pleuritic effusion or empyema in the anterior and internal portion of the left pleura, solidified aneuris-

mal tumor, abscess descending from the neck, or hypertrophy of the heart.

Partial suppression of the sounds of the heart may result from functional debility, or fatty degeneration of the organ; and from the use of certain medicinal agents in excessive doses; for example, bromide of potassium, digitalis, and nicotine. Impairment or suppression of the impulse may be the consequence of fatty transformation or extreme dilatation of the heart and thinning of its walls; and both the impulse and the sounds may be impaired, or apparently suppressed, by displacement of the heart from the anterior wall of the chest, through the agency of an emphysematous or a cancerous lung, or a mediastinal tumor.

Increase of the area of percussion-dulness in the precordium may be due to any of the causes above enumerated, as capable of giving rise to enlargement of the area over which respiratory sound is suppressed; to which may be added, localized pneumonic solidification of the anterior portion of the left lung.

Displacement of the apex of the heart may be caused by liquid or aeriform effusion into either pleura, enlargement of the liver, hepatic or perihepatic abscess, aneurism, mediastinal tumor, or diaphragmatic hernia of the stomach.

Pericardial *frottement* may be simulated by pleuritic friction-sound developed in the anterior portion of the left pleura; by an endocardial-murmur; and by emphysema of the anterior mediastinum, as shown by Dr. Hudson.* It may, in every case where present, be distinguished from the two former phenomena; but it is wanting in most cases of liquid effusion into the pericardium to an amount capable of giving rise to morbid signs.

Da Costa maintains that suspension of sound during some of

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* *Dublin Quarterly Journal of Medicine*, vol. viii., p. 241, August 1st, 1849. In this interesting case there was extensive dulness of the precordium ; but it does not appear that this was in any degree qualified, as might be expected, by mediastinal resonance. A *frottement* of a " crackling character," synchronous with the sounds of the heart, but strikingly distinct from them, and loudest to the left of the sternum, was detected at the base of the heart. The diagnosis of pericarditis was made by the author of the paper, a most accomplished stethoscopist. On examination of the body, the pericardium and heart were found to be perfectly sound ; the areolar tissue of the anterior mediastinum was distended with air, and extended somewhat to the left, in front of the pericardium. A blood-clot contained in the right auricle was likewise partially occupied by air-bubbles.

the beats of the heart constitutes the only means of distinguishing pericardial from localized left pleural *frottement.** No doubt, occasional intermission of the friction-sound would warrant a *presumption* in favour of pleuritis; but much stronger evidence to the same effect might be deduced from the localization of the friction-sound *externally* to the apex-point, and its total absence elsewhere in the precordium, as pointed out by Dr. Stokes, independently of the coexistence of respiratory-sound in the seat of friction, where pleuritis is the cause of the phenomenon.

Extension of precordial dulness upwards, in a degree not readily accounted for by reference to other conceivable causes, should raise a suspicion of pericardial effusion. It may, however, depend upon pneumonia limited to the superior lobe of the left lung, carcinoma of the lung or mediastinum, or circumscribed empyema.

The elements for a positive diagnosis of liquid in the pericardium are of a compound character, and are rarely all present in any one case; hence the difficulty, in many instances, of arriving at a positive opinion.

Localized bulging of the precordium, engaging an increased extent of surface, dull on percussion, devoid of respiratory sound and movement, and of vocal thrill, without displacement of the heart, the cardiac sounds being faint and distant,† and associated or not with *frottement* and irregular action of the heart, might be held to warrant the diagnosis of liquid in the pericardium. Obliteration of the intercostal spaces in the precordium, with visible undulation synchronous with the movements of the heart, variation of the seat of apex-pulsation according to the posture of the body, and displacement of the liver downwards, should be regarded, when present in the same case, as confirmatory of the diagnosis of pericardial effusion. But, although of the utmost value diagnostically, these signs must not be looked for in the majority of such cases, because their presence is certainly the exception. I cannot say that I have

* *Medical Diagnosis*, third edition, 1870, p. 357.

† Dr. Da Costa (*opus citat.*, p 355) justly remarks, that where the sounds of the heart are masked in the precordial region by liquid effusion into the pericardium, the second sound is distinct, and even sharp, along the sternum and in the course of the ascending aorta.

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derived much assistance from the alleged triangular definition of the area of extended precordial dulness in these cases. Indeed, I believe this test to be practically unavailable, from the difficulty of tracing the outline of a dull surface of limited extent, with such accuracy as to distinguish between one of a triangular and one of an ovoid figure. It must be borne in mind, as eminently pertinent to this inquiry, that disproportionate extension of the horizontal line of precordial dulness inferiorly, which is alleged to be distinctive of effusion into the pericardium, may be due to enlargement of the liver or spleen.

Extension of localized dulness considerably to the left of the point of apex-pulsation, if associated with absence of respiratory sound of any kind to the same extent, would assist materially in establishing the identity of liquid effusion into the pericardium, and would suffice to distinguish it from hypertrophy of the left ventricle on the one hand, and from localized solidification of the adjacent portion of the left lung upon the other; and, similarly, limitation of dulness to the antero-inferior portion of the chest, equally in the erect and recumbent posture of the body, would practically exclude liquid effusion in the left pleural cavity.

Unfortunately for the value of the former of these alleged diagnostics, the apex-pulsation is *not* to be felt where the pericardium is distended with liquid, save in the few cases in which the apex is fixed by adhesion to the precordium. The heart may, no doubt, be brought into contact with the anterior wall of the chest by causing the patient to assume the prone or stooping posture; but in this position of the body it would be difficult, or even impossible, to determine the extent of dulness, with a degree of accuracy sufficient to constitute it a legitimate basis for differential diagnosis. Nor, indeed, does it seem necessary; for the distinction between left ventricular hypertrophy and hydropericardium, even based upon the positive signs of the former affection exclusively, can never be difficult.

Doctor Gee says, " The first extension of percussion-dulness occurs at the base of the heart, where the great vessels enter, and where the pericardium hangs loosely round them, and is most distensible."* But the pericardium is in a state of ten-

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* *Auscultation and Percussion*, 1870, p. 247.

sion physiologically at all points. Under the influence of the inflammatory process, however, it becomes relaxed, and equally so, throughout its entire extent. As a matter of fact, I have never met with an example of primary dulness at the base of the heart from pericardial effusion, and cannot admit such, either on physiological or clinical grounds, till definite proof of its occurrence shall have been furnished. On the contrary, I have found that in the progress of adhesion or liquid effusion, persistence of friction-sound at the base, even for several days after it had ceased to be audible elsewhere, whether with or without increase in the area of precordial dulness, is the rule. Such is likewise the opinion of Da Costa, who says that when the stage of serous effusion has set in, and the pericardium contains liquid even in considerable quantity, "it is not uncommon for the ear still to recognize the murmur at the base of the heart, and around the origin of the great vessels."*

Recurrent pericarditis, by which is meant a second or subsequent invasion of the disease, is characterized by a group of symptoms dependent upon the nature and extent, not only of the present, but likewise of the previous attack. If complete adhesion be amongst the consequences of the primary affection, the secondary accession will not be characterized by friction-sound or fremitus. Partial adhesion in a previous attack, however, does not preclude friction-phenomena in one of a later date, but at a point of surface distinct from that where adhesion has actually taken place.

The recurrence of such phenomena, therefore, amounts to proof that antecedent pericarditis has not issued in universal cohesion of the parietal and visceral pericardium. In such cases, also, liquid effusion can be only partial, and superficial dulness must be proportionately limited.

Doctor Moxon is of opinion, and I quite agree with him, that disorganization of the epithelium, which is the necessary result of adhesion of the pericardium, indisposes the serous structure to recurrent inflammation.† When such inflammation

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* *Opus citat.*, p. 353.

† "Lectures on Analytical Pathology ;" lecture viii. *Medical Times and Gazette,* November 12th, 1870.

occurs, therefore, the cause may be presumed to have been of a more than ordinarily exciting character, and the results, both as to symptoms and inflammatory products, in excess of those usually witnessed. Hence the aggravated character of the symptoms, and the great thickness of the pericardium, by which recurrent pericarditis is generally characterized.

If, in a previous attack, the substance of the heart has been invaded by inflammation, and consecutive degeneration of its fibres has made progress in the interim, the symptoms of the recent accession will be of a formidable character *quoad* the circulatory and respiratory functions. In such a case there is likely to be failing pulse, vertigo, sensation of sinking and oppression at the precordium, palpitation and threatened syncope on making even the slightest effort, oppression of breathing, and congestion and œdema of the lower limbs. Epigastric fulness and tenderness, and slight albuminuria, but without lowered specific gravity of the urine, are amongst the consecutive but late symptoms of recurrent pericarditis, where tissue degeneration of the heart has preceded. Manifestly, in such a case, the heart would barely be competent to maintain the circulation at the standard of feeble health; hence, when further embarrassed by the products of renewed inflammation, or debilitated by a secondary extension of that process to its muscular substance, the heart is no longer capable of performing its functions, and the consequences of its partial but progressive failure are amongst the symptoms of the recurrent disease. Antecedent endocarditis, by disorganizing the valves, and thereby entailing upon the heart the consequences of valvular lesion, namely, hypertrophy and dilatation, may be likewise the indirect cause of a formidable group of symptoms having reference to partial failure of the circulation, in a primary or secondary attack of pericarditis. It is noteworthy that, whilst a second attack of endocarditis is of exceedingly rare occurrence, secondary pericarditis is by no means uncommon; and further, that the secondary disease is usually not different from the primary, either as to site or essential character, the apparent difference depending upon the reduced power of the heart by preceding inflammation.

Under the designation of *latent pericarditis*, Drs. Law and

Stokes have described a form of the affection which is not announced by symptoms; the physical signs, however, being well pronounced.

Doctor Law has met with examples of it in connexion with disease of the lungs, both acute and chronic;* and Dr. Stokes has witnessed it as a complication of the essential fevers, and in diffuse inflammation, phlebitis, and erysipelas.†

In the preceding connexions, the symptoms of pericarditis are *masked* by the constitutional disturbance attendant upon a major disease.

Chronic pericarditis, according to Corvisart, is obscure in origin and difficult of diagnosis.‡ Laennec says it is always general, occupying the entire inner surface of the pericardium, and that the surface is of a redder tint than in the acute form, owing to the presence of a multitude of minute vascular points.§

It is most frequently the consequence of slow convalescence or protracted dissolution, from an attack of the disease in the acute or subacute form. It may, however, be primary, and in that case it is usually associated with chronic visceral disease, *par excellence*, that of the kidneys. When primary it is usually latent as to symptoms, being announced only by physical signs, which are, however, most commonly of an exaggerated character. In this form of the disease, likewise, the fibrin effused upon the surface of the pericardium rarely undergoes organization, remaining loosely attached to the surface, or floating in the liquid contents of the sac as white filamentous shreds. This is the form in which pericarditis is most frequently associated with hypertrophy of the heart, because of the concomitant disease of the kidneys to which both are due; or with hypertrophy and dilatation, the consequence of valvular disease coeval with the pericarditis, and the direct source of the increase in the volume and capacity of the heart. The structural changes in the pericardium, constituting in such cases the first and the most striking

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* *Proceedings of the Pathological Society, passim.*
† *Diseases of the Heart and the Aorta*, p. 338.
‡ Hebb's translation.
§ *Mediate Auscultation*, Forbes' edition, p. 661.

alteration to be met with in a *post mortem* examination of the heart, and affording as they do, on the authority of Hope, a full and satisfactory explanation of the increased volume of that organ, have been admitted by pathologists as in every instance the primary lesion, and the starting point of the other organic changes discovered. The state of the kidneys has been rarely inquired into, and valvular disease of the heart, when detected, has been regarded as a contributory cause of hypertrophy, but of secondary importance in its influence.

Doctor Barclay states that in twenty-two out of sixty-one, instances of valve-disease, there was disease of the kidneys with albuminuria.* No doubt, albuminuria is of frequent occurrence in valvular disease, indeed almost universal in the advanced stages; but it is in the majority of cases the result of simple congestion of the kidneys, the specific gravity of the urine being rarely under 1030.

Circumscribed pericarditis, as indicated by localized friction-sound, may be presented under two opposite conditions, as stated by Doctor Stokes;† one, in which, apparently, from failure or arrest of inflammatory action due to an unknown constitutional influence, or to early and effective treatment, the exudation of lymph is limited to one or more circumscribed spaces, over which for a variable, but sometimes for a considerable period, the sounds of friction may continue to be heard. In such cases the seat of friction usually corresponds to the sides of the ventricles, the most frequent site being, according to my experience, the third and fourth intercostal spaces of the left side, midway between the nipple-line and the sternum. In such cases, likewise, adhesion, or serous effusion rarely takes place. Secondly, a condition in which, after general pericarditis, and, it may be, liquid effusion, adhesion has proceeded to all but complete obliteration of the pericardial sac; in such case, friction may continue to be audible over a small space, generally at the base, but occasionally at the apex of the heart, for an indefinite period, and may mislead an incautious auscultator into making the diagnosis of hæmic murmur or valvular lesion.

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* *Medico-Chirurgical Transactions*, vol. xxxv., November, 1851.
† *Diseases of the Heart and Aorta*, p. 17.

The observance of the rules already laid down for the distinction of pericardial *frottement* from endocardial murmur, to which I beg to refer the reader, will always, however, render such a mistake in the highest degree unlikely to occur.

" *Milk spots* " are, I believe, due to circumscribed and aborted pericarditis of a subacute character, and identical with that form of arachnitis in which thickening and opacity of the membrane constitute the only visible alterations of texture resulting from even fatal inflammation; the epithelium is perfect in the true " milk spot."

Corvisart denied the inflammatory origin of these patches, and attributed them to fibrinous deposit *beneath* the pericardium.*

Laennec asserted that there was no example of albuminous exudation on the attached surface of a serous membrane; adding that pseudo-membranous exudations are almost invariably products of inflammation. He regarded the " white spots " as due to inflammation of the pericardium, and as consisting of deposit *on* its free surface.† Such cases I would regard as veritable examples of circumscribed and arrested pericarditis, yielding friction-sound during life, and presenting, when examined after death, a rough surface due to deposited fibrin, which, in many instances, may be removed from them in form of flakes. In neither of these particulars does the true white spot answer to the description just given; it is not associated with abnormal sound of any kind, and its surface is invariably smooth and glistening, and formed of tesselated epithelium.

Louis agreed with Laennec in regarding the " white spots " as the result of pericarditis, but limited to the visceral layer exclusively.‡

Mr. Paget says that in connexion with these spots there is usually, to some extent, adhesion of the pericardium; from which circumstance he concludes they are of pericarditic origin. In the greater number of the examples which have fallen under his notice, adhesion did not exist in the situation of the spots, a circumstance due, as he supposes, to the separa-

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* Hebb's translation, p. 40.
† *Mediate Auscultation*, by Forbes, p. 661.
‡ *Mémoires ou Recherches Anatomico-Pathologiques*, 1826.

tion of the surfaces by liquid during the progress of adhesion of the neighbouring surfaces. The adhesions consist of filaments between the aorta and superior cava, or the aorta and pulmonary artery; between one of these vessels and the parietal pericardium, or between one of the spots and the latter surface. Finally, there may be only vestiges of adhesion in the form of "small pearly granules." Their most common site is the anterior surface of the right ventricle; less commonly they are found on its posterior surface, or on the right auricle; still more rarely on the left ventricle; and least frequently on the left auricle. Their average width is half an inch; their thickness varies from half a line to two lines; and, according to the degree of inflammation, and the depth to which the membrane is engaged, the exuded lymph will occupy its surface or its substance. When the organization of the exuded lymph is complete, a new epithelium is formed upon its surface.*

In forty-five, out of one hundred and fifty-six bodies, Bizot has observed the "white spot" on the right ventricle. He regarded it as due to thickening of the pericardium from some unknown cause. In his opinion it is three times more common in males than in females; rarely, if ever, seen before the age of seventeen; and not common before forty years.†

Doctor John Taylor has found "white spots," or patches, in a nearly similar proportion of the bodies examined in University College Hospital; viz., in one out of every four.‡

Finally, Copland attributes the "white spots" to pericarditis.§

Thus, it would seem to be the opinion of the majority of writers, that the patches usually seen upon the heart in advanced age, and designated "milk spots," or "white spots," are due to antecedent inflammation.

In this opinion, however, as above stated, I by no means share.

Endo-pericarditis, as may be inferred from what has preceded,

* *Medico-Chirurgical Transactions*, vol. xxiii., 1840.
† *Recherches sur le Cœur et le Système Arteriel chez l'Homme.*
‡ *London Medical Gazette*, new series, vol. i., 1845.
§ *Dictionary of Medicine.*

is a much more serious affection, and involves more grave consequences, both immediate and remote, than simple pericarditis.

Doctor Sibson has found endocardial complication of rheumatic pericarditis in one-half of his cases (three out of six).* It has occurred in a proportion of somewhat more than one-third of my cases. The compound affection is characterized by the symptoms and signs of the more grave element of it; namely, the endocarditis. It is, accordingly, associated with more decided respiratory distress, and greater derangement of the circulation. Respiration is quick and panting; the pulse is very rapid, ranging usually from 120 to 140, sharp and abrupt; the heart's action tumultuous, and the cervical arteries visibly pulsating, whilst those of the limb do not seem inordinately excited.

Doctor Stokes lays much stress upon this symptom in connexion with pericarditis; and, without expressing a positive opinion, would seem to regard it, when recent, and associated with febrile action not due to inflammation or irritation of the encephalon, as diagnostic of the complication of endocarditis.† There are five different conditions, according to him, in which localized violent pulsation of the carotids may be exhibited; namely, concussion of the brain, as mentioned by Sir Astley Cooper, permanent patency of the aortic valves in the early stage, cerebritis, acute endocarditis, and exophthalmic goitre. By the absence of pyrexia, and by the positive symptoms of the disease, which cannot be mistaken, the last named affection will be readily excluded. The history of concussion of the brain, the impairment, or total loss, of consciousness, and the subsequent vertigo and vomiting, sufficiently distinguish this condition. Patency of the aortic valves will declare itself by its pathognomonic sign of basic diastolic murmur, transmitted through the aortic arch, but *not* into the arteries of the neck; and endocardial inflammation complicating pericarditis, may, though it rarely does, actually assume the form capable of giving rise to this sign; in which case carotid-pulsation would be attributable to a twofold cause; namely, one of a general, and one of a specific nature. The symptoms of cerebritis usually regarded as specific,

* Address in Medicine, British Medical Association, August, 1870.
† *Opus citat.*, p. 52.

may be readily mistaken for the cerebral manifestations of cardiac inflammation, and the actual existence of the physical signs characteristic of the latter condition can alone be relied on for the differential diagnosis.

An endocardial murmur of a determinate character is heard within a brief period subsequent to invasion. This murmur, though in a measure masked by the more superficial, harsh, and much louder pseudo-murmur of pericarditis, may always be distinguished from the latter, firstly, by its rhythm either coinciding with, or replacing one of or both, the sounds of the heart, or immediately preceding or succeeding these sounds: in other words, by the strict association of the murmur with the sounds of the heart, as estimated by time ; a character by which it is readily distinguishable from the sounds of pericarditis, which are eminently arhythmical. Secondly, inasmuch as the endocardial murmur most often associated with pericarditis is that of mitral reflux, it must be, and actually is, audible where the pericardial friction-sound rarely reaches; viz., the left axilla and left scapular region. Thirdly, the murmur of endocarditis is at this early stage always soft and blowing in quality, unless when rendered musical, as occasionally, but very rarely, happens for a brief period, by the vibration of a flake of fibrin attached by one of its extremities to the free margin of the valve ; whereas attrition-sounds are invariably rough and harsh. And, lastly, a valvular murmur, as stated by King Chambers, is perceptible to the ear raised a few lines from the stethoscope. Not so an attrition-sound.

I have never met with an example of "systolic murmur at the base of the heart from the pressure of effused lymph," as alleged by Andral,* without, however, the production of any *post mortem* evidence in support of the assertion. No case has come under my notice in which, with false membrane engaging the roots of the great vessels, a basic murmur, when present, was not legitimately attributable to other, more generally recognized, and certainly more common, causes of this phenomenon; viz., lesion of the valves, atheroma of the root of the aorta, thrombus, or spanæmia.

* *Anatomie Pathologique.*

Bouillaud held that *bruit de soufflet* in pericarditis occasionally depends upon thrombus in the heart, and, "conceivably," upon the pressure of liquid in the pericardium.[*]

I have neither met with an example of the latter kind, nor can I conceive a degree of liquid pressure in the pericardium sufficient to resist the impetus of the column of blood escaping by the aorta or the pulmonary artery, which would not, at the same time, of necessity so impede the movements of the heart as seriously to interfere with the circulation.

Thus, it would seem, theoretically, that a murmur due to the cause alleged should be associated with partial failure of the heart and its consequences.

Hope maintained that excessive force of ventricular contraction is competent, of itself, to cause murmur.[†]

Law holds a similar opinion. [‡]

I have never heard a murmur attributable to this cause, and should feel great difficulty in admitting its occurrence.

Flint is of opinion that pericarditis is "almost always, if not invariably, associated with endocarditis;"[§] from which it follows that, in his judgment, endocardial murmur is an all but invariable concomitant of pericardial *frottement*; and that, when present, it owes its origin, in the great majority of cases, to inflammation of the valves.

I cannot admit either of these propositions in the form in which they are presented. The association of endo- with pericarditis is, according to my experience, the exception; although, judged by the numerical proportion in which endocardial murmur is likewise present, it might be otherwise regarded.

Doctor Stokes states that, in his experience, the endocardial murmur is audible at a later period of the compound disease than pericardial *frottement*, and suggests the inquiry whether, in a certain proportion of these cases, the endocardial murmur may not be due to impeded action of the heart, through a close and firm adhesion of the pericardium already established.[||]

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[*] *Traité Clinique.*
[†] *The Diseases of the Heart.*
[‡] *Dublin Journal of Medical and Chemical Science*, vol. viii., 1834.
[§] *A Treatise on the Principles and Practice of Medicine*, 1868, p. 304.
[||] *Opus citat.*, p. 34.

I cannot regard this hypothesis as probable, save to the extent to which it implies possible antecedent myocarditis, and consequent softening aud yielding of the walls of either ventricle.

Doctor John Taylor has found valvular disease in about one-half of the cases of rheumatism examined by him; but the greater number of these examples he believes to be of old standing. He is of opinion that the frequency of acute endocarditis in rheumatism has been greatly overrated, and that it does not by much, if at all, exceed that of pericarditis.* Of sixty-two examples of valve-disease reported by Dr. Barclay,† only twenty-three were associated with a history of acute rheumatism, and, even amongst these, in three the lesion did not appear to be of rheumatic origin. Hence, less than one-third of the cases were rheumatic, and in reference to twenty-six, rheumatism was positively denied. The distribution of these cases was as follows:

	Rheumatic.	Non-rheumatic.
Disease affecting both sets of valves (mitral and aortic)	12	10
Disease affecting mitral valve alone ...	7	11
Disease affecting aortic valve alone ...	1	5

The author's cases:

	Rheumatic.	Non-rheumatic.
Disease affecting both sets of valves ...	2	1
Disease affecting mitral valve alone ...	2	1
Disease affecting aortic valve alone ...	0	0

There was another case (No. 7, the woman Nolan) in which temporary mitral systolic-murmur existed, but which I regarded as thereby excluded from the category of valvular lesions.

Where the two affections are not simultaneous in occurrence, and this is the rule, the pericarditis is usually the primary morbid element. In cases characterized by progressive engagement of the structure of the heart, or extension of inflammation by contiguity, from the investing to the lining membrane, or vice versa, the muscular substance of the organ must have been involved at some period of the disease. Hence the symptoms characteristic of myocarditis enter into the category of those actually

* Loco citat.

† Medico-Chirurgical Transactions, vol. xxxv., November 11th, 1851.

present, and being attributed in all probability to the primary inflammation of the investing or of the lining membrane of the heart, as the case may be, give rise, so far, to a vitiated symptomatology. I have no doubt that in this way must be accounted for many of the symptoms erroneously attributed to simple pericarditis, or endocarditis, such as irregular or intermittent pulse, delirium, and convulsions. Of the single valvular lesions met with in association with pericarditis, mitral inadequacy is by far the most common ; and, of those of compound character, mitral inadequacy, with aortic obstruction and inadequacy, holds the highest place numerically.

But the development of an endocardial-murmur in the early period of convalescence from pericarditis, is by no means proof of antecedent endocarditis, or of the actual existence of valvular lesion. .

Doctor Stokes says : " The mere occurrence of murmur, even though immediately consequent on pericarditis, is not necessarily indicative of progressive valvular disease." He adds : " Thus much is certain, that the occurrence of murmur following pericarditis should not necessarily lead to the diagnosis of valvular disease in the ordinary acceptation of the term," for " it may occasionally be found that the murmur after existing for a period more or less extended, disappears, leaving the sounds of the heart in the natural condition, and the patient remains free from symptoms of valvular disease."* He suggests, in explanation of temporary murmur in such cases, endocarditis not followed by deposit, atony of some portion of the muscular fibre, or alteration of the orifices, and consequent maladaptation of the valves by tonic spasm of the heart.

I have repeatedly witnessed examples of this kind (*e.g.*, Case 7, Mrs. Nolan,), and am of opinion the murmur is caused by atony and expansion, at the acme of ventricular systole, of a portion of the wall of the left ventricle, and consequent elevation of one or both segments of the mitral valve through the connecting papillary muscles and tendinous chords. Such murmurs, when not manifestly hæmic in origin, are always mitral and systolic; they cease more promptly under tonic treatment, in-

* *Opus citat.*, p. 2.

cluding iron, than under any other; and rarely, under such treatment, exceed a few weeks in duration.

I cannot, therefore, agree in opinion with Dr. Latham that the cessation of an endocardial murmur denotes, in all cases, removal of newly deposited matter.*

The symptoms characteristic of *myo-pericarditis* have been already incidentally mentioned; they are such as might have been anticipated from the operation of any cause acting directly upon, and enfeebling the centre of the circulation.

Myocarditis, as a primary affection, is of exceedingly rare occurrence; indeed, it may be stated that, independently of the knowledge of the affection derived from experience of it by extension from an inflamed pericardium or endocardium, nothing is positively known in regard to its symptomatology. It most frequently commences by extension of acute inflammation from the pericardium to the contiguous or outer layers of the muscular substance of the heart, and may proceed no farther, engaging only the external portion of the organ. I believe this is of common occurrence in acute pericarditis of an aggravated character, the really alarming symptoms of which, or those having reference to irregularity and threatened failure of the circulation, are attributable to inflammation of the muscular rather than of the serous structure of the heart. Thus it is that in the progress of pericarditis, the action of the heart and the pulse may become irregular or intermitting. Syncope may be threatened, or, on slight exertion, may actually occur, and the impulse of the heart, notwithstanding its tumultuous action, may be feeble, and the first sound masked or ill pronounced.

If the disease be arrested at this stage, fatty degeneration, but of a slow and progressive character, of the outer muscular substance of the heart is likely to ensue; and thus it is, as I believe, that the greater number of such examples are produced. The inflammatory process extending inwards may engage the entire substance of the heart, and, if not immediately fatal, may give rise either to suppuration, or to fatty degeneration of its structure, according to the activity of the preceding inflammation. The early stage of inflammation of the heart, or that of acute conges-

* *Lectures*, vol. i., p. 149.

tion, is characterized anatomically by dark red discoloration of the portion of its substance engaged ; the muscular structure, so altered in colour, is soft, and easily torn.   After a period of variable length, it becomes light in colour, or assumes a yellow tint, according to the rapidity of the disorganizing process.   If suppuration set in, the muscular substance becomes infiltrated with pus, light in colour, and lacerable in the highest degree ; minute abscesses may be formed in various portions of its substance, and may lead to ulceration.

The process of fatty transformation of the muscular substance of the heart is much less rapid, and the result of more chronic inflammatory action.

Fatty metamorphosis of the heart may owe its origin to endocarditis, and commence at the internal surface of one or more of the chambers, thence slowly progressing outwards, and ending either in suppuration or in fatty degeneration, to a greater or less extent, of the substance of the heart.   The characteristic features of such a case would be those of endocarditis, modified by symptoms of partial and momentary failure of the heart.   Thus, there would be precordial oppression, quick and tumultuous action of the heart, and hurried respiration   To these would succeed quickly, it might be within a few hours, irregularity and intermission of the heart and pulse, vertigo, and temporary loss of consciousness, paroxysmal dyspnœa, and collapse of the features.

From this alarming condition partial restoration may be effected, to the extent of enabling the patient to resume his ordinary duties; but, fatty transformation of the substance of the heart, whether immediate or consecutive to purulent infiltration, once started, is progressive, and leads inevitably to death at a more or less remote period, according to circumstances influencing the rate of its advance.

Those who are obliged to labour bodily or mentally to obtain a livelihood, more readily succumb to this process, owing to the twofold cause of rapid molecular disintegration, and imperfect nutrition of the heart.   Those who, on the contrary, are in a condition of life which enables them to have repose, and at the same time suitable nutriment, may live to a comparatively advanced

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age, even though they be actually the subjects of fatty transformation of the heart commencing in endo- or pericarditis.

The triple combination implied by the compound title *endo-myo-pericarditis* is of comparatively rare occurrence. When met with, it is characterized by a corresponding combination of signs and symptoms; viz, the valvular murmur of endocarditis, the friction-sound of pericarditis, and the symptoms of failure of the heart, already detailed, by which myocarditis may be confidently diagnosed Amongst the latter symptoms, those having reference to the cerebrum, and presented under the forms of delirium, convulsions, and coma, may be appropriately discussed here.

Delirium is mentioned by Corvisart[*] and Bertin[†] as a symptom of acute pericarditis, without, however, any statement of opinion as to its cause. Bouillaud likewise mentions, as a symptom of pericarditis, "delirium, usually slight and momentary, sometimes violent," and occasionally followed by convulsions.[‡]

Andral gives the particulars of a case of pericarditis in which delirium was a prominent symptom. A young woman, after a brief illness, became delirious; there was improvement and return of consciousness, twitching of the muscles of the face, occasional tetanic spasm of the upper extremities, return of delirium, coma, and death. Soft exudation was found on the pericardium, with some bridles of lymph extending from surface to surface, and, in the sac, some ounces of green flocculent serum[§]

In this case the effusion present in such small quantity could not have been the cause of the cerebral symptoms; whilst its colour and quality indicated that the substance of the heart was involved in the inflammatory process, although, in appearance, it is described as "presenting nothing morbid." He quotes a similar case from Rostan,[‖] and attributes the head symptoms, in both cases, to a "sympathetic affection of the brain." by which

[*] *On Diseases of the Heart*, Hebb's translation.
[†] *Traité des Maladies du Cœur*, 1824, p. 252.
[‡] *Traite Clinique des Maladies du Cœur*, 1835.
[§] *Clinique Medicale*, vol. 1, second edition, 1829, observation viii.
[‖] *Recherches sur le Ramollissement du Cerveau*, p. 233

he manifestly means to convey failure of the circulation in that organ.

Doctor Latham mentions two cases of pericarditis in which the cerebral symptoms were so pronounced as to resemble those of inflammation of the brain,* and Hope includes delirium and convulsions amongst the symptoms occasionally witnessed " in the last stage," and regards them as the result of " cerebral congestion, and the circulation of venous blood;"† in other words, as symptoms of asphyxia due to slow failure of the heart and congestion of its right cavities. Dr. Sibson details the case of a man, aged twenty-eight, a free liver, who, on the evening of the sixth day of acute rheumatic pericarditis, became violently delirious, and presented symptoms generally resembling those of delirium tremens. He died on the eighth day. Both layers of the pericardium were found covered with honeycombed lymph. No mention is made of the condition of the heart substance.‡

Sir T. Watson gives the particulars of four cases of rheumatic arthritis, in which the heart was affected and symptoms suggestive of cerebral inflammation also existed. The only pathological changes, however, exhibited by the brain in these cases, were slight serous infiltration of the pia mater, and partial effusion into the lateral ventricles.§ Dr. Bright details the case of a young man who, on the sixth day of an attack of acute rheumatism, became excited and delirious; the symptoms became more urgent, and it was found necessary to put the patient under restraint. At the end of three weeks he died. The brain was found perfectly healthy, but the pericardium and the endocardium exhibited evidence of recent and acute inflammation‖ Dr. G. Burrows records an example of death in a state of delirium after seven days' illness. The brain was, to all appearance, unaltered, but the pericardium was covered with recent lymph, and, over a small space, with concrete pus.¶ Dr. Todd furnishes a no less striking example of a similar kind. A young woman,

* *London Medical Gazette*, vol. iii , p. 209.
† *Opus citat* , p. 156.
‡ *Lectures on the Principles and Practice of Phys e* fourth edition, vol. ii , p. 303
§ Address in Medicine, British Medical Association, 1870.
‖ *Medico-Chirurgical Transactions*, vol. xxii
¶ *Disorders of the Cerebral Circulation.*

some days ill of rheumatic fever, was seized with delirium, which was succeeded by convulsions, coma, and death. The brain and its membranes exhibited no inflammatory product, and were even paler than normal, whilst the pericardium was covered with recently exuded lymph.* Dr. Fuller is of opinion that the cerebral symptoms exhibited in the course of acute rheumatism, are due to the toxic action of the rheumatic virus on the brain † Trousseau regards cerebral manifestations in acute rheumatism as the result of a "neurosis," and maintains that they may be primary; preceding, or even replacing the rheumatic affection of the joints or of the heart, and meriting the designation of "cerebral rheumatism,"‡ according to him. Todd§ and Fuller are of opinion that an organic poison, the product of a morbific process associated with rheumatism, constitutes one and the principal factor in the production of the cerebral phenomena, but that another and no less important factor is a morbid impressionability of the cerebro-spinal nerve centres, hereditary or acquired.

The presence, within the cranium, of the pathological products of acute inflammation; viz., lymph and pus, has been observed by Watson and Fuller. The association, however, is exceedingly rare, and may have been due to a simple coincidence. In acute cerebro-spinal meningitis, the occurrence of articular pain and tumefaction is not uncommon; but, the order of manifestation, and the general character of the symptoms of this disease, would preclude the possibility of confounding it with articular rheumatism.

Da Costa says that in pericarditis the thoracic symptoms may be in abeyance, and others, indicative of gastric or cerebral irritation or inflammation, may take their place; these latter being, in his opinion, often due to the poison of rheumatism, or to that of Bright's disease.¶

Flint mentions, as occasionally witnessed amongst the symp-

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* *Lectures on Delirium and Coma*
† *On Rheumatism and Rheumatic Gout*
‡ *Clinical Medicine*, vol. i, p 534, Sydenham Society's edition
§ Lumleian Lectures, *Lancet*, 1852.
   Loco citat.
¶ *Medical Diagnosis*, third edition, 1870, p. 358.

toms of pericarditis, paroxysms of maniacal excitement, alternating with alarming dreams, and starting from slumber.*

Doctor King Chambers thinks that delirium in rheumatism indicates vascular and nervous depression, requiring stimulants, tonics, and opium; and that it is more frequently symptomatic of pericarditis or pneumonia than not.†

The different opinions held as to the pathogenesis of cerebral symptoms, associated with rheumatic inflammation of the heart or its membranes, may be conveniently presented in synoptical form.

Partial failure or derangement of cerebral circulation, through inflammation and consequent debility of the heart ..	{ Andral, Watson, Burrows.
Rheumatic poison acting on susceptible nerve-centres ..	{ Trousseau. Todd. Fuller. Da Costa.
Cerebral congestion and circulation of venous blood .	Hope.
Vascular and nervous depression (dynamic?)	King Chambers.

It would appear, therefore, that pericarditic delirium and convulsions have been attributed by writers to two different and nearly opposite pathological conditions; namely, defect of arterial circulation in the brain through failure of the left heart, and the circulation through that organ of a blood vitiated by an organic poison elaborated within the body itself, or by retained excreta.

I have not witnessed well pronounced delirium or convulsions in connexion with pericarditis. Occasional wandering of mind, and partial incoherence, easily dissipated by sharply addressing the patient, and such as is commonly witnessed in essential or inflammatory fever, have frequently come under my notice in the first and second stages of pericarditis. In the stage of serous effusion, accompanied with engorgement of the cervical veins, lividity of the features, and failure of the heart, I have likewise witnessed incoherence. Mental obscuration is usually manifested in partial asphyxia from copious effusion, under the

* *A Treatise on the Principles and Practice of Medicine*, Philadelphia, 1868, p 804, *et sequent.*

† *Lectures, chiefly Clinical*, third edition, 1864, p. 142.

form of somnolence and muttering, contraction of the pupils, and slowness in collecting the thoughts; on being roused to the effort, however, the patient is capable, after a little hesitation, of giving a connected answer.

In the former case, the slight mental derangement present is due, as in ordinary febrile excitement, to rapid disintegration of tissue and imperfect excretion; in the latter it is the consequence of failure of the *right* side of the heart, and venous engorgement of the brain.

Syncope has been noticed in connexion with pericarditis by Bertin, who witnessed it in two cases out of thirty-six,[*] and mentions faintness, failure of the pulse, and anxiety, as amongst the ordinary symptoms.[†]

Louis had not an example of syncope amongst his four cases, and he denied that it was an essential symptom of pericarditis.[‡] Neither have I witnessed an example, unless fatal syncope, by which several of the cases that had proceeded to the stage of liquid effusion terminated, be admitted into this category.

Traumatic pericarditis presents no peculiar features by which it is distinguishable from the ordinary or idiopathic form of the affection, save those imparted to it by the general and local effects of the mechanical lesion. Dr. Stokes gives the particulars of a case of this kind resulting from a gun-shot wound, a charge of small shot having been lodged in the superficial structures of the precordium; after the lapse of a few days the symptoms and signs of pericarditis were exhibited, the latter being characterized by a number of distinct seats of friction-sound of various intensity and duration. The patient recovered, and no opportunity was therefore presented for determining the precise depth or character of the injury; but, it is presumed that a few pellets had actually penetrated the pericardium, and been so many independent centres of inflammation.

Flint records a very remarkable example of the same kind. A person in a state of intoxication swallowed a set of false teeth, which lodged in the œsophagus, and effected a communication

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[*] *Traité des Maladies du Cœur*, 1824, observation lxiii., p. 289.
[†] *Opus citat.*, p. 270.
[‡] *Mémoires ou Recherches Anatomico-Pathologiques*, 1826.

with the pericardium by ulceration. The specimen is preserved in the Bellevue Hospital, New York.[*]

Amongst the *intrinsic complications* of pericarditis are included tubercular deposition in, and cancerous and calcareous transformation of, the pericardium. Of the two former, mention is made by Bonetus[†] and Hasse;[‡] of the latter, examples are quite numerous, and several have been already adverted to. The process of deposition, or of tissue-transformation, would seem in every case to engage the false membrane or neoplastic structure only, at least in the first instance.

The *extrinsic complications* of pericarditis may be classified under two heads, constitutional and local. Those of a general or *constitutional* character most frequently met with are Bright's disease in its various forms; the exanthematous fevers, especially scarlatina; diffuse inflammation; pyæmia; gout; and, preeminently, rheumatism.[§]

Doctor John Taylor states that of thirty-five cases of aggravated pericarditis, one-third nearly were associated with Bright's disease, and two-thirds with acute rheumatism. He has found acute pericarditis as a secondary inflammation in the advanced stages of Bright's disease, in the proportion of one in ten. [||]

Doctor Mayne returns five out of his eleven cases of pericarditis as rheumatic.[¶] Eight of my cases, thirteen in number, were of rheumatic origin. Out of a total of seventy-four cases of acute and subacute rheumatism reported by Dr. Sibson, six only were affected with pericarditis; and of twenty-three cases of

[*] *A Treatise on the Principles and Practice of Medicine*, 1868.

[†] *Sepulchretum.*

[‡] *Pathological Anatomy.* Sydenham Society, 1846, p. 116.

[§] In the strict sense, the term "complication" is not applicable to the above-mentioned diseases in their relationship to pericarditis; they constitute, in each instance, rather the original disease. To an author, however, treating of pericarditis as a distinct and independent entity, it is convenient, even though its secondary importance, pathologically and therapeutically, in any given category, be admitted, to regard concurrent or coexistent disease, of whatever kind, as a complication. Indeed, in regard to any disease under special consideration, all other diseases having any relationship to it, might be conveniently classified under the three heads of *causal, casual,* and *consecutive* complications. In this sense the diseases mentioned in the text would belong to the first of these three classes.

[||] *London Medical Gazette*, new series, vol. i., 1845.

[¶] *Dublin Journal of Medical and Chemical Science*, vol. vii.,1835.

acute gout returned by the same author, only one was affected with pericarditis; and even this one was of a transient character *
I have already adverted to the opinion of Trousseau, to the effect that the pericarditis of scarlatina is likewise of rheumatic origin, being due to a rheumatic complication of that disease frequently witnessed under the form of arthritis, rather than to the poison of scarlatina.† This is a very questionable doctrine, even to the extent of assuming the existence of the twofold poison. It is, at best, only an hypothesis, which is not susceptible of proof or open to refutation, and manifestly not necessary to a full explanation of the facts. Intercurrent inflammation and swelling of various structures and organs are common in scarlatina; and serous membranes, other than the pericardium, are frequently implicated. Dr. Law remarks, with truth, that where pericarditis arises from, or in connexion with, diffuse inflammation, the effusion is likely to be purulent.‡

Amongst the extrinsic *local* complications of pericarditis may be mentioned pleuritis, pneumonia, pleuro-pneumonia, and hepatitis. In most of the cases of pericarditis published by Corvisart, one or other of these complications was present,§ most frequently pleuro-pneumonia and diaphragmatic pleurisy. Gastritis has been likewise mentioned by Corvisart as a frequent complication; but, manifestly, on insufficient evidence, as the only symptom pointing to gastritis indicated by him was epigastric tenderness, a symptom now generally attributed to pericarditis itself, but in many instances due to hepatic congestion.

Inflammation of the lungs accompanied pericarditis in more than one-half of Dr. Latham's cases, and endo-pericarditis in more than two-thirds; whilst of endocarditis alone the proportion of cases in which pulmonary inflammation existed was only one in nine. ||

Graves details an example of pericarditis complicated with, and dependent upon, hepatic abscess, as already quoted.¶

* Address in Medicine, British Medical Association, 1870, pamphlet, p. 24
† *Clinical Medicine*, vol. i., p. 397.
‡ *Reports of Pathological Society of Dublin*, vol. i, p 42
§ Hebb's translation, p. 6, *et sequent.*
|| *London Medical Gazette*, new series, vol. i, 1845.
¶ *Clinical Medicine*, vol. ii., p. 233.

Doctor John Taylor gives an example of pericarditis by extension of inflammation from the pleura in one instance, and from the liver and diaphragm in another.[*]

Acute pericarditis is a disease of *early* and *middle life*. I have repeatedly seen it in early childhood, but never in persons over forty years.

Louis mentions a fatal case of pericarditis in a child of seven months, which had been related to him by Boudard,[†] and is of opinion that the periods of life at which pericarditis is most common, are between seventeen and thirty, and between fifty and seventy years, respectively. I have never seen a case of pericarditis within the latter period, except in the chronic form, and as a complication of advanced renal disease.

Out of fifty-five cases of pericarditis given by Hache, only six were over forty years of age; and Fuller declares that pericarditis is of much more frequent occurrence in youth than in advanced age.[‡]

Chronic pericarditis, on the contrary, is most common in the aged; because this is the period of life in which chronic visceral disease, of which it is usually a complication, especially renal disorganization, is most prevalent.

In regard to the relative proportion of the *sexes* affected with pericarditis, most authors agree that the majority are of the male sex.

Out of one hundred and seven cases given by Louis,[§] only twenty-seven were females; and in those published by Bizot, seven in number, one only was of that sex.[||]

Bouillaud reports only six females out of a total of thirty-four cases of pericarditis[¶]

Hache estimates the proportion as one female to four males.[**]

Of thirteen cases given by the author in the preceding table (p. 360), nine belonged to the male sex.

[*] *Loco citat.*
[†] *Mémoires ou Recherches Anat. Pathol.*, p. 292.
[‡] *Archives Générales de Médecine*, tom. ix, November 1835, p. 328.
[§] *Diseases of the Chest*, p. 513.
[||] *Loco citat.*
[¶] *Traité Clinique des Maladies du Cœur.*
[**] *Loco citat.*, p. 119.

Sibson's six cases of pericarditis were in males, and all under thirty-four years of age.[*]

The preponderance of males amongst those affected with pericarditis may, therefore, be regarded as established. This will probably be satisfactorily explained by reference to the more laborious occupations and greater exposure to vicissitudes of weather and changes of temperature, of males of the working classes, who are most frequently the subjects of pericarditis, whether rheumatic or renal.

Fuller maintains that females are more liable to rheumatic pericarditis than males,[†] and Dr. Barclay declares that this is likewise true of other cardiac complications of rheumatism.[‡]

There is no divergence of opinion, however, between these writers and those previously quoted, because they allege only what is admitted by all; namely, a larger per centage of pericarditis, relatively to the number affected with rheumatism, amongst females than amongst males.

Rheumatic pericarditis is fatal in the proportion of about one in six cases, according to Fuller[§] and Louis. I have had only one death in eight cases (see table, p. 360).

The *prognosis* in pericarditis should be determined rather by the complications than by the disease itself. Simple pericarditis is rarely fatal. In patients of strumous cachexia, however, serous effusion into the pericardium, causing death by paralysis of the heart, has occasionally taken place. The opinion of Corvisart, to the effect that acute and chronic pericarditis are always fatal, the subacute form being only a degree less unfavourable, will now find few if any subscribers, and is explicable only by the imperfect state of the science of diagnosis at the time he wrote. It will be remembered that at the date in question the principles of mediate auscultation were still unknown, and that, consequently, those cases of pericarditis which were of a sufficiently formidable character to make themselves manifest by *symptoms* so pronounced as not to admit of misinterpretation,

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[*] *Loco citat.*
[†] *Opus citat.*
[‡] *Medico-Chirurgical Transactions,* vol. xxxv
[§] *Opus citat.*

were alone diagnosed. The diagnosis of pericarditis, in the acute form, was based upon the general effects of liquid effusion into the pericardium; and, in the chronic form, upon the symptoms indicative of adhesion of the pericardium with hypertrophy and dilatation of the heart. That Andral laboured under the same disadvantage may be inferred from the following declaration: "And if no pain exist in the region of the heart, there only remains to recognize the pericarditis, the dull sound of this region."[*]

Flint truly remarks that the prognosis depends upon the complications; and that it is most unfavourable in Bright's disease, and in cases of copious effusion rapidly poured out. In regard to the issue of pericarditis in its milder form, I unreservedly subscribe the following rule of practice laid down by Dr. Stokes: "While any signs continue, he (the patient) must be carefully watched; for, in certain cases, a sudden change occurs, and the disease is converted from an apparently trivial and latent affection into a more severe form."[†] The prognosis is less favourable in those of weak constitution than in the strong, and in childhood than in adult age, for the reason previously stated.

It is supposed by some writers that the *season* exercises a definite and perceptible influence on the prevalence of pericarditis. Thus, Hache declares that nearly one-half (eighteen out of thirty-eight) of the cases recorded by him occurred in the last four months of the year, and only one-fourth in the first four; viz., from January to May.[‡] Inasmuch as the prevalence of pericarditis is mainly regulated by that of acute rheumatism, of which it is usually a complication, the season and the climate most favourable to the latter will obviously be those in which pericarditis will be most prevalent.

Doctor Fuller shows that within the first four months of the year, rheumatism is slightly more common than during the corresponding periods of the middle and terminal portions.[§]

I have found rheumatism more prevalent during the cold fogs

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[*] *Clinique Médicale*, vol. i., p. 38, foot note.
[†] *Opus citat.*, p. 45.
[‡] *Loco citat.*
[§] *Rheumatism, Rheumatic Gout, and Sciatica*, third edition, 1860, table iv., p 44.

of November and December than at other periods of the year; and, in like manner, its most frequent complication, pericarditis. In all countries and seasons a moist atmosphere has been found favourable to the production of rheumatism and pericarditis.

The *treatment* of pericarditis resolves itself into that of the disease in its acute, chronic, and recurrent forms respectively.

At the outset of acute pericarditis in the young and vigorous, Bouillaud and Hope urge the abstraction of blood, *coup sur coup*, by means of the lancet, and, locally, by leeches or the cupping-glass. A daily bleeding from the arm of twenty to thirty ounces, followed by an equal loss locally, for two or three consecutive days, would not have been considered by them as an excessive amount in the treatment of the first stage of acute pericarditis, in a young subject of previously unimpaired health.

I have never had to treat a case of pericarditis that seemed to require, or even to warrant, so vigorous a plan of treatment. I prefer, in every case, local abstraction of blood by means of leeches; ten, twenty, or thirty, according to the urgency of the case, and the strength and constitutional tolerance of the patient. This measure might, if necessary, be repeated once or oftener, and should be invariably succeeded by the application over the leech-bites of a warm poultice. The necessity for a repetition of leeching should be determined by the impulse, rhythm, and sounds of the heart, and by the sensations of the patient as referred to the heart. If the impulse be strong and abrupt, the rhythm regular, the sounds, especially the first, normal or sharp, and a sensation of oppression or pain at the precordium be still experienced by the patient, leeching should be repeated to such extent as may be deemed necessary to effect the object held in view; namely, to arrest the progress of inflammation and subdue these urgent symptoms. I have never used the cupping-glass for this purpose in the first instance, as I should apprehend the possible occurrence of unpleasant consequences, such as syncope, from the shock of its application. I can, however, see no objection to the use of a dry cup for the purpose of abstracting blood from lancet-punctures or leech-bites. Immediately blood in sufficient quantity has been abstracted by a first leeching, my invariable practice in acute cases is to administer mercury in

small and frequently repeated doses.   I usually direct that half a grain of colomel, with one grain of James' powder, shall be given in powder or pill every hour.   For the James' I substitute Dover's powder if there be a tendency to diarrhœa; and if it actually exist I give grey powder or blue pill with opium, in equivalent doses at corresponding intervals.   I have found this mode of administering mercury, commonly known as that of Dr. Law, in acute sthenic inflammations, more rapidly efficacious, and much more manageable, than that of giving it in doses of ten to twenty grains twice or thrice daily, as proposed and prac-tised by Dr. Johnson and sanctioned by Dr. Graves.   Admin-istered in this manner, I push the use of mercury to slight salivation, and am satisfied when the imprint of the teeth, with white and elevated lines of union, appear upon the inner surface of the cheeks; when the gums present a swollen, soft, and spongy appearance, and when coppery taste is experienced, and a slight drivelling of saliva at the angles of the mouth takes place.   These results are usually attained, in cases of ordinary impressiona-bility, within a period of twenty-four hours, and simultaneously a noticeable improvement in the patient's general condition takes place ; the pulse has descended in frequency, and become relaxed, the surface temperature reduced, and, perhaps, the skin moist, breathing less rapid and embarrassed, and the feeling of oppression at the heart less urgent, or entirely abolished.

A saline aperient should be administered at the outset, if the bowels be not actually over relaxed.   I usually prescribe a seidlitz powder with a drachm of syrup of ginger; or two drachms of the acid tartrate of potash, with a drachm of tinc-ture of ginger, and two ounces of the infusion of senna.

When the symptoms of improvement above described have appeared, under the treatment just sketched, the use of wine, in moderate doses, and frequently repeated, should be immediately commenced, with a view to obviate such a state of cardiac debility and relaxation of the coronary vessels as might favour the occurrence of serous effusion into the pericardium.   It should be given in doses of at least one ounce every second hour; and, above this minimum, the quantity should be regulated by the impulse and sounds of the heart, and the quality of the

radial pulse. If the cardiac impulse be of moderate force, the first sound distinct and clear, and the second sound single, the pulse at the wrist being full and monocrotic, the minimum allowance of wine may be deemed sufficient. But if, on the contrary, the impulse of the heart be feeble, the first sound muffled and indistinct, and it may be, the second sound double, whilst the pulsations of the radial artery are small, feeble, or dicrotic, and perhaps occasionally lost, wine in much larger doses, and given every hour, would be demanded. To those who have been addicted to the free use of ardent spirits, I allow, in preference, whiskey or brandy in equivalent quantity.

Whilst treating of this subject, Dr. Stokes remarks: "In truth, it may be said, that no man is fit to treat general disease, or local inflammation, especially its secondary forms, until he has conquered that fear of stimulants, which a long course of erroneous teaching has instilled into his mind."[*]

Doctor Todd has been charged, or rather his memory, with having, in his posthumous work edited by Dr. Beale,[†] advocated the use of alcoholic stimulants in the treatment of acute disease to an unwarrantable extent, and set thereby a dangerous example to less judicious practitioners. I can only say that, having read this lecture with the greatest care and attention of which I am capable, I failed to discover in it any justification for this charge. Dr. Todd advocates the *rational* use of stimulants, somewhat as I have above recommended them; and after a full and able exposition of their physiological action, the circumstances under which they are admissible, and the benefits derivable from their use, he says, "Alcohol is a form of aliment appropriate to the direct nourishment of the nervous system, and to its preservation. Taken carefully, it increases the animal temperature; it also strengthens the action of the heart; and when administered under proper circumstances it reduces the frequency of the pulse.

"You must give enough to keep up animal heat, and to protect the tissues without embarrassing the stomach, and without allowing the fumes of alcohol to be perceived in the breath.

"Alcohol may be employed in all those diseases in which a

* *Diseases of the Heart and Aorta*, p. 90.   † *Lectures*, 1861, lecture xvi

tendency to depression of the vital power exists, and there are no acute diseases in which this lowering tendency is not present.

" Delirium is a symptom of enfeebled and contaminated nutrition of the brain. It is to be looked for in all exhausting diseases, and in all acute maladies accompanied by high fever.

" When alcohol is given, as I have recommended, it calms the nervous system, promotes tranquil sleep, from which the patient may be easily roused, and averts delirium.

" The rule of its administration should be 'a little and often,' from two to sixteen drachms, more or less dilute with water or other bland fluid, and repeated at such intervals as his state of vital power may seem to demand "

From the preceding extracts, it will be manifest that Dr. Todd was by no means an advocate for the free and indiscriminate use of alcohol in acute disease, as has been alleged. On the contrary, the indications for its use, and the rules for its administration both as to quantity and mode, which he lays down, are those which guide the most enlightened practice of the present time.

In regard to the effect of alcohol upon the temperature of the body, however, exact experiment has recently established, contrary to the inferences of Dr Todd, that it is either neutral, or positively depressing, according to the actual state of health of the subject of experiment. Thus, Dr. Parkes has shown that it in no degree affects the temperature of a healthy body, and does not increase the body weight, or affect the elimination of nitrogen by the bowels or kidneys; whilst Professor Binz has experimentally proved that alcohol sensibly and continuously lowers temperature in fever.

Clinical experiment and observation have decisively sanctioned the enlightened use of alcoholic stimulants in most acute diseases. But, in dealing with those acute inflammatory affections in which the mass of the blood is brought into immediate contact with the inflamed surface, as in endocarditis, endarteritis, and phlebitis, alcoholic agents, though occasionally demanded, should, as a rule, be eschewed.

An opiate sedative, consisting of a grain of the watery extract, or fifteen minims of Batly's sedative, should be given at

night, with the view of insuring sound sleep, by which the excitement of the heart is allayed. I find opium, in some one of its forms, the most suitable and efficacious hypnotic in such cases: it is usually well borne, even in large doses.

The diet should be bland and nutritious, consisting of beef-tea, chicken-broth, arrowroot, etc.; and, after acute febrile action has subsided, solid food in small quantity, such as a chop, or the breast of a chicken, may be allowed, provided the tongue be moist. For drink, saline beverages should be given, such, for example, as the so-called "imperial," consisting of an ounce of bitartrate of potash dissolved in a quart of water, flavoured with lemon-juice, and sweetened to the taste; milk and soda water in equal proportions; or the natural alkaline waters of Vichy, Vals, Ems, or Apollinaris.

When the acute inflammation has been subdued, as indicated by subsidence of acute febrile symptoms, I substitute for the previous lowering treatment some tonic medicine, such as decoction of cinchona with iodide of potassium, in doses of three grains of the latter to an ounce of the former; or infusion of gentian with bicarbonate of soda; and, in every case, wine or whiskey at this stage, in quantity determined by the general condition and previous habits of the patient.

Doctor Stokes sanctions a single bleeding from the arm in a case of uncomplicated pericarditis in the earlier stages, but prefers abstracting blood locally by means of relays of leeches, from ten to twenty, repeated three or four times within the twenty-four hours if necessary, and succeeded by warm poultices. He advocates the administration of calomel at the same time, and recommends that it shall be given in large doses after the method of Graves.* The use of calomel with opium is recommended also by Fuller,† and, indeed, by most modern authors.

Opium has been freely administered in acute pericarditis, with a view to allay the excitement of the heart, at the same time that topical abstraction of blood is being effected. This practice, which certainly has its advantages in the treatment of acute pericarditis associated with great nervous excitement, pal-

* *Opus citat*, p. 84.
† *Rheumatism, Rheumatic Gout, and Sciatica,* third edition, 1860.

pitation, and feeble circulation, as in anæmic and hysterical females, has been lately advocated very strongly by Dr. Frederick J. Farre,* who reports most favourably of it. He gives eleven cases of cure, under a plan of treatment consisting in the administration of one grain of opium every sixth hour.

Doctor Chambers, likewise, strongly recommends opium in the treatment of both acute rheumatism and rheumatic pericarditis.† He no less decidedly disapproves the use of mercury in the treatment of the former, on the ground that, by increasing relatively the proportion of fibrin in the blood, it predisposes to pericarditis.

In the second stage of acute pericarditis, after the subsidence of febrile action, I would, in addition to the treatment above indicated, namely, iodide of potassium and alkalized bitters, make use of counter-irritation in some form, with a view to keeping up a derivative action from the heart; and with the same object in view, I would reapply leeches at any period of this stage, should the evidence of renewed inflammation, especially a return of precordial pain, be presented. Probably the best form of counter-irritant in such a case would be the vesicating collodion, or an ordinary blister brushed over with this fluid, the object being to effect rapid as well as complete vesication. A warm poultice continuously applied over the blister will accelerate its action, but its weight cannot, in many cases, be borne. As a substitute for a blister, I have frequently used in this stage a mercurial and opiate plaster, consisting of half an ounce of the ordinary mercurial plaster of the Pharmacopœia, with ten grains of opium, spread upon strong paper and laid over the heart, as first recommended by Dr. Lionel Beale. I give a preference to this application, when much nervous excitement of the heart exists, with loud and grating friction-sound.

The use of digitalis may be demanded at this period of the disease. If tumultuous, and at the same time irregular action of the heart occur, digitalis, in doses of five to ten minims of the tincture, given every third hour, will be found efficacious in lowering the rate, restoring the rhythm, and increasing the

* *St. Bartholomew's Hospital Reports*, vol. ii.
† *Lectures chiefly Clinical*, 1864, p. 126.

strength, of the heart's action.* Colchicum and bark have been recommended in this stage; but, Stokes truly remarks that these medicinal agents should be given with caution where fever or cardiac excitement exist.

In a comparatively small proportion of cases, pain of a paroxysmal character, and resembling that of angina pectoris, occurs. In such cases it will probably be found that the other symptoms and signs lead to the diagnosis of myo-pericarditis. In the treatment of this affection, Dr. Latham has found opium in comparatively large doses to act most beneficially as an ano-dyne and sedative. In combination with chlorodyne I have found it of the utmost value. Ten to fifteen minims of chlo-rodyne with an equal quantity of Battley's sedative, given in an ounce of water, sufficing usually to insure relief from urgent pain and cardiac distress.

In the third stage I rarely give digitalis, because of the existence of a mechanical impediment to the movements of the heart; the exceptions are cases in which, other measures having failed to dissipate a serous accumulation in the pericardium, I would try its effects as a diuretic, before having recourse to the *dernier ressort*, paracentesis.

In the treatment of liquid collections in the pericardium I have no faith in blisters, and rely mainly upon diuretics and hy-dragogue purgatives. Of medicines of the former class, acetate of potash in half drachm doses, given, every second hour, with a drachm of spirits of juniper in an ounce of compound decoction of scoparium, is that to which I usually give a preference; and as a purgative, I give the compound powder of jalap in doses of half a drachm to a drachm, with two to four grains of calomel, accord-ing to the age, strength, and susceptibility of the patient. The al-lowance of liquids should be, at the same time, restricted to the smallest possible amount; indeed, the patient should be urged to allay rather than satisfy thirst, by frequently rinsing the mouth, or sucking a morsel of ice. During the entire of this period wine should be given, in quantity determined by the force and quality of the pulse.

* I would not venture the application of powder of digitalis to a blistered sur-face over the heart, as recommended by Bouillaud.

In many cases, the best directed and the most active medicinal measures will fail to dissipate serous collections in the pericardium. In such cases, the sac is usually distended, the heart is impeded in its movements, the pulse is irregular and failing, and the patient's life is in imminent jeopardy. At any moment, under these circumstances, death may suddenly occur by syncope on the patient's making the slightest muscular effort, such as that involved in sitting up to drink, or even turning in bed. One alternative alone now remains to the physician, and one which, if it is to save life, must be promptly put in execution; namely, *paracentesis pericardii.*

Senac advocated this operation in the treatment of hydropericardium, and recommended, as the best instrument for the performance of it, the trocar and cannula. He has not, however, adduced even a single case in his own practice to exemplify the benefit derivable from the operation.

Corvisart believed " that the advantage to be derived from this operation will rarely counterbalance the danger which attends it ;"[*] but should it be decided upon, he gives a preference to the bistoury for its performance.

Desault actually essayed the operation; but, having proceeded upon a wrong diagnosis into which he and his colleagues were led, namely, that of mistaking a circumscribed pleuritic effusion for hydro-pericardium, he did not enter the pericardium, as was proved by examination of the body after death.

Doctor F. A. Aran has successfully performed the operation, and subsequently injected iodine into the pericardium : the patient recovered.[†] The solution injected consisted of equal proportions of tincture of iodine and water, with one-fourth part, by weight, of iodide of potassium.

Successful performance of the operation, followed by recovery of the patient, has been likewise recorded in the practice of Jobert de Lamballe,[‡] Romero of Barcelona (two examples),[§]

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[*] *Opus citat.,* p. 52.

[†] *Bulletin de l'Académie de Médecine,* tom. **xxi**, p. 142 ; and *Dublin Hospital Gazette,* December 1st, 1855.

[‡] *Trousseau's Clinical Medicine,* Sydenham Society's edition, vol. iii., p 371.

[§] *Dictionnaire des Sciences Médicales,* vol xl., 1819.

Wheelhouse,* and Karawagen.† The procedure has been prac-
tised, successfully as to operation but unsuccessfully in regard to
result, by Trousseau, Larrey, Schuh, Heger, Romero, Wheelhouse,
and Karawagen, as exemplified in the following table:

TABLE IV.

Operator.	Successful.	Unsuccessful.	Erroneous Diagnosis.	Mode and Site of Operation.	Death.	Recovery.
Desault	—	1	1	Bistoury, between fifth and sixth ribs	1	—
Trousseau... ...	1	—	—	Bistoury, below nipple	1	—
Jobert de Lamballe	1	—	—	Bistoury and trocar in fifth intercostal space	—	1
Larrey	—	1	1*	Ditto	1	—
Schuh	1	—	—	Trocar and cannula, between third and fourth ribs, near sternum	1	—
Heger	1	—	—	Trocar and cannula; two operations	1	—
Romero	1	—	—	Bistoury and scissors, between fifth and sixth ribs	—	1
Romero	1	—	—	Ditto	—	1
Romero	1	—	—	Ditto	1	—
Bowditch	—	—	—	Bistoury	1	—
Roux	—	1	1			
Aran	1	—	—			
Aran	1	—	—	Bistoury and capillary trocar, in fifth intercostal space	—	1
Aran	1	—	—	Ditto	—	1
Wheelhouse ...	—	—	—	Ditto	—	1
Wheelhouse ...	1	—	—	Capillary trocar, in fifth intercostal space	1	—
Karawagen ...	1	—	—	Not stated	—	1
Karawagen ...	1	—	—	Ditto	1	—
18	13	3	3		9	7

* Uncertain. No P. M.

* *British Medical Journal,* 1868.
† *British and Foreign Medico-Chirurgical Review,* vol. xii., July, 1841.

Thus, out of a total of eighteen cases, thirteen were successful in regard to the immediate object sought; namely, the removal of liquid from the pericardium; three were in this respect unsuccessful, and in each instance failure was due to an error of diagnosis; death followed in nine instances, and recovery in seven. In eleven instances the bistoury was the principal instrument used, and in three, the trocar and cannula. In no instance, to my knowledge, has the pneumatic aspirator been used in the performance of this operation.

I have never yet performed the operation, nor had it done by another; but, on two occasions, at least, I have had reason to regret the omission of its performance. The circumstances which warrant, and even demand, the operation, as it seems to me, are the following.

If the presence of copious liquid effusion in the pericardium be indubitable, as determined by the evidence previously insisted upon (p. 387), and there exist, at the same time, urgent dyspnœa, small and failing pulse, œdema of the lower limbs and face, and venous engorgement of the neck, medicinal agency having failed to disperse the liquid collection or ameliorate the general condition of the patient, in my judgment the chance presented by the operation should be promptly given. There should be, of course, no misgiving as to the diagnosis where so formidable an operation is undertaken; yet it is not always easy to arrive at certainty on this head. Stokes says: "The diagnosis between mere dropsy of the pleura and the pericardium is not always free from difficulty."* And, on the same subject, Trousseau declares that, "Notwithstanding the comparative certainty which has in our day been attained in the diagnosis of effusion into the pericardium, the diagnosis is still sufficiently difficult to leave room for the physician being deceived; and the special form of the dulness in pericarditis very different from the rounded dulness due to hypertrophy of the heart, is a very uncertain sign."† He then gives, by way of illustration of the difficulty mentioned, two examples of erroneous diagnosis of pericardial effusion by most competent physicians; namely, Dr. Vigla of the Hotel Dieu, and himself, in one of which the opera-

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* *Opus citat.*, p. 92, foot note.   † *Opus citat.*, vol. iii, p. 383.

tion was actually proceeded with to the stage of exposing the pericardium before the mistake was detected; and in the other, although, according to his judgment, the operation was justifiable, he hesitated, and postponed its performance; meantime the patient died. In the former of these cases great dilatation of the heart alone existed; and in the latter, exaggerated hypertrophy with adhesion of the pericardium.

Extended area of percussion-dulness, dependent upon simple dilatation of the ventricles, would, however, in the great majority of cases, be readily distinguishable from that due to liquid in the pericardium, by the sharp, clear, and well defined character of the sounds of the heart; whereas, in examples of liquid effusion, the sounds are masked, ill defined, and heard as from a distance.

Exaggerated dilatation is, moreover, usually associated with pulmonary emphysema and congestion of long standing, together with systemic venous engorgement and anasarca. It must, however, be admitted that the supervention of pericarditis with slight serous effusion in such a case, or the occurrence of simple passive dropsy of the pericardium, even to the amount of a very few ounces, so commonly associated with it in the advanced stage, may lead the most accomplished auscultator to diagnose copious liquid effusion exclusively.

For the differential diagnosis of cardiac hypertrophy, as usually presented, I would rely upon the prolonged and masked character of the first sound, as contrasted with the sharpness, clearness, and abnormal loudness of the second; the heaving character of the impulse, and the displacement of the point of apex-pulsation In hypertrophy, moreover, the sense of resistance to percussion in the seat of dulness is less than where it is caused by liquid effusion.

As in the example given by Trousseau, a thin stratum of liquid, the product of antecedent pericarditis or the result of passive effusion, occasional complications of cardiac hypertrophy from renal disease, interposed between the parietal pericardium and the heart, may so effectually mask the impulse and apex-pulsation as to render an accurate diagnosis difficult or even impossible. Such cases must be exceedingly rare, as I have met with only a single example.

In the progress of cardiac hypertrophy of renal origin, if the patient be not prematurely cut off by one or more of the other complications of renal disease, fatty degeneration of the heart will certainly ensue ; and, as a necessary consequence, its action will be so much enfeebled, and its sounds so masked, that the criteria just mentioned as between hypertrophy and hydro-peri-cardium, utterly and signally fail, and still more probably if slight effusion be superadded. In one such case (Case 29, Miss T.), I made the diagnosis of copious serous effusion into the pericardium, and on examination of the body after death, I found not more than two ounces of liquid in that cavity.

The site of puncture should be " of election," and determined mainly by two circumstances ; namely, the situation of maxi-mum dulness and most distant cardiac sounds, on the one hand, and the absence, at the point contemplated, of the faintest approach to impulse, on the other. The fourth or fifth intercos-tal space, from one inch to two inches to the left of the sternum, has been usually selected for puncture, and consistently with the observance of the conditions above mentioned, will be found the safest and most convenient point to select. It should always be borne in mind, in selecting a point for puncture, that the heart may be attached to the pericardium at any part of its entire superficial area, and hence no absolute rule can be laid down, irrespectively of the two conditions above mentioned, for the guidance of the operator in the selection of the site of punc-ture. The avoidance of the internal mammary vessels renders it necessary to puncture not less than one inch from the edge of the sternum.

I would not contemplate the trephining of the sternum, as proposed by Laennec, nor the division of the eighth costal carti-lage of the left side at its junction with the sternum, as recom-mended by Larrey. The former plan of proceeding would involve grave dangers other than those properly appertaining to the operation, such as emphysema of the anterior mediastinum, and through it, of the entire body ; osteo-phlebitis ; and caries of the sternum. The latter mode of operation would have the effect of weakening the entire framework of the chest, and would

by no means insure the object contemplated ; namely, the avoidance of the pleura.

For the performance of the operation, I should certainly give a preference over all other instruments to the pneumatic aspirator ; by the use of which the entrance of air into the pericardium and pleura would be effectually prevented, the escape of liquid into the cavity of the pleura avoided, and if, by misadventure, a wound were inflicted on the heart during the operation, it would be of so trivial a character, as to lead, in all probability, to no serious consequences.

Doctor Sibson recommends that a fine exploring trocar and cannula should be introduced obliquely upwards, either to the left of the xiphoid cartilage, or at the anterior part of the fifth intercostal space, and the fluid removed by means of a syringe applied to the extremity of the cannula.* Thus he recognizes the advantages conferred by the application of the principle of suction in the performance of this operation, and I doubt not that he would now adopt, in preference, the modern and more perfect instrument for the application of this principle; namely, the pneumatic aspirator.

In persons exhibiting the scorbutic diathesis in an aggravated form, as exemplified in sailors before the improvement of the navigation law, by which a penalty is attached to the non-shipment of fresh food and lime juice preparatory to a long voyage, the great serous cavities are liable to sudden and profuse extravasations of blood, which, acting as a foreign body, becomes a cause of acute inflammation of the most unfavourable augury.

Scorbutic pericarditis, so excited, is characterized by the suddenness of its invasion, and by the accompanying dyspnœa. The heart-sounds are faint and remote, and the area of precordial dulness is rapidly and greatly extended; patients so affected rarely survive beyond the third day.

Karawagen, whose experience of the disease during an epidemic of scurvy among the sailors at Kronstadt, has been considerable, states that of sixty men who died of scurvy, thirty exhibited pericarditis of this form; twenty-two pleuritis; two

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* *Medico-Chirurgical Review*, July, 1854.

both pericarditis and pleuritis; and one peritonitis. The peri-
cardium was thrice its natural size, and contained a bloody
exudation amounting to four or five pints.   No breach of
vascular wall could be detected.   The heart was reduced to
one-half of its natural size, and both it and the parietal peri-
cardium were covered with a red mould-like substance, which
was readily removed by scraping with the edge of a knife.   The
substance of the heart was soft and fatty, and the endocardium
was tinted red by imbibition; the ventricles were compressed
and empty   Paracentesis was performed in two of these cases,
in one of which it completely succeeded, the patient having
been restored to perfect health after an interval of five months,
although three and a-half pints of liquid had been removed from
the pericardium.*

Of this form of the disease, the differential diagnosis of which
would manifestly depend upon the general evidence of scor-
butus, the treatment should be mainly antiscorbutic, and should
the physical evidence of sudden and copious effusion be pre-
sented, and accompanied by urgent dyspnœa and threatened
failure of the heart, paracentesis would be indicated, as afford-
ing the only chance of saving the patient's life, and should be
promptly performed.

Wounds of the pericardium are attended with considerable
pain; although the heart itself is insensible to mechanical
injury, according to Harvey, Ollenroth, and Bamberger. Georges
Fischer gives fifty-one examples of wound of the pericardium,
out of which there were twenty-two recoveries.†   *Traumatic
pericarditis* is always formidable, because of the all but neces-
sary entrance of air into the sac, and consequent suppuration.
The most active treatment should be promptly instituted, with a
view to subduing the inflammation.   Thus, leeching, mercury,
and opium, should be at once directed, in proportion to the
activity of the fever and the urgency of the symptoms.

Tuberculosis and *cancer* of the pericardium have been men-

* "On Scorbutic Pericarditis and Pleuritis," *British and Foreign Medico-Chirur-
gical Review*, vol. xii , July, 1841.

† *Des Plaies du Cœur et du Péricarde, Archives Générales de Médecine* (Review),
vol. i., p. 612, May, 1869.

tioned by Bonetus,[*] Laennec,[†] Hasse,[‡] Stokes,[§] and Niemeyer.[‖] Tubercle, when present, is usually found in the pseudo-membranous product of chronic pericarditis, and associated with tuberculosis of the lungs, and with strumous enlargement of the mediastinal and bronchial glands. The tubercle is most frequently presented in the miliary form; but it may undergo caseous transformation, rarely, however, proceeding to suppuration.

Carcinoma of the pericardium is usually a secondary formation, and consecutive to cancer of the anterior mediastinum, or of the pleuræ; it is, in most cases, of the soft or medullary kind, and associated with turbid and flaky effusion into the pericardial sac.

But when the pericardium is the seat of cancer, that disease rarely succeeds ordinary pericarditis as a consecutive pathological transformation of exuded lymph; in this respect it differs pathologically from tubercle, which rarely appears within the pericardium, save as a new formation in false membrane.

The following cases will serve to illustrate the clinical history, pathology, and treatment of pericarditis.

CASE I —*Chronic Endocarditis with Mitral Inadequacy; Acute Pericarditis with Effusion; Double Frottement; Death.*

Anne L., aged nine years, admitted into hospital, March 26th, 1866. Has had articular rheumatism, with swelling. A fortnight since was attacked with pain in the left side, accompanied with shortness of breath. At the date of admittance, the child was pale and fidgety; pulse 132, but regular; respiration 72. A double or "to-and-fro" friction-sound was audible over the lower part of the sternum, and a single systolic murmur at the apex. The latter was *slightly musical,* and audible likewise at the angle of the left scapula. Ordered, an opiate and mercurial plaster (Opium, gr. v, Emplast. hydrarg., ℥iv, spread upon strong paper'

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* *Sepulchretum.*

† *Auscultation Médiate,* vol. ii., p. 658.

‡ *Pathological Anatomy,* Sydenham Society's edition, 1846, p. 116.

§ *Opus citat.*

‖ *Text-Book of Practical Medicine,* 1869, vol. i., p. 395.

over heart. ℞. Pulv. Ipecac. co., Pulv., Hydrarg. c. creta, aa. gr. ij. M. Ter die sumend.

March 29th. Pulse 126, steady, and of good volume; she is less choreic; "to-and-fro" sound audible at lower end of sternum, but soft; continue.

April 2nd. Dulness on left side to level of second rib, with absence of *frottement;* "to-and-fro" sound heard faintly, but more distinctly during pressure with the stethoscope. Patient fidgety, very weak and faint; pulse 126, and feeble. Wine to ℥ij

4th. Pulse 120, of good volume; precordial dulness of much less extent; "to-and-fro" sound now audible at apex; remarkably loud at base, and diffused generally over front of chest, but with greater distinctness upwards and to right side; continue.

5th. Pulse 132, and weak; respiration 54; increased extent of precordial dulness, with faint fremitus; "to-and-fro" sound not audible over apex, and less loud at base. Sinapism over heart, and continue. For several days past she has had herpetic eruption on lips.

6th. Pulse 120; respiration 42, irregular and suspirious; no friction audible over apex, but loud friction over base; no *frémissement.*

7th. Sat up yesterday; pulse 120, and much stronger; respiration 30; friction-sound diminishing, and audible only at base; precordial dulness less in extent; continue.

9th. Pulse 132; friction less distinct; systolic murmur audible at angle of left scapula; has bronchitis. ℞ Iodid potass., gr. v, in decoction of cinchona thrice daily; a stimulating liniment to be applied over the precordium.

11th. Is very weak; pupils dilated; pulse 138; no cardiac impulse to be felt; there is dulness on left side from second rib downwards, and no friction is audible. Ordered, a blister to precordium, and tincture of digitalis, ℳv, in an ounce of decoction of scoparium every third hour; increase of stimulants.

20th. Precordial dulness is now of normal extent; "to-and-fro" sound is no longer heard, but over the apex of the heart a systolic bellows-murmur is heard with all its previous intensity; pulse 120, and full; tongue clean; appetite good. Patient has

been out of bed for the last three days, and during that time has been taking sulphate of quinia in gr. ½ doses thrice daily.

30th. Discharged to-day, restored to health in all respects, with the exception that, at the apex of the heart and in the left back, there is still audible a loud systolic bellows-murmur, precordial dulness normal; sleep and appetite unexceptional, and for several days past she has been taking exercise about the ward and corridor

May 14th. She was readmitted to-day in a state of great debility, unable to stand without support; cardiac impulse was heaving; a double murmur was heard at the apex, and very indistinctly in the course of the aorta; pulse weak; cough, with expectoration; bilious vomiting. Blister to precordium, wine, chloric ether and spirit of camphor, lime water and milk.

17th. Is much relieved; stomach settled.

19th. On the evening of this day she became suddenly weak and agitated; breathing became embarrassed and gasping; she spat some blood, and continued to have fits of partial syncope every ten or fifteen minutes; the extremities were cold, and there was scarcely any pulse at the wrist; face pale and anxious; respiration loud, harsh, and rapid, and associated with bronchial râles on the left side, which was the only one admitting of examination, owing to the difficulty of moving the patient; she was unable to speak. Sinapism over heart, and a dessert-spoonful of brandy every second hour.

21st. She seemed moribund.

22nd. To my astonishment I found the patient, at my morning visit, cheerful; no longer suffering from dyspnœa or hæmoptysis; but the heart's action, as before, was associated with double murmur, and respiration was loud, harsh, and irregular.

23rd. Precordium prominent, and dull to an increased extent; capillary venous congestion over lower part of chest on both sides; cardiac impulse barely perceptible, and sounds scarcely audible. Blister over heart.

29th. From the date of last report patient was in the agony of death, up to 2 o'clock on yesterday (28th) when she died on being lifted out of bed contrary to my direction. The body and face

had become œdematous ; she had a teasing cough, and continued
to expectorate small quantities of florid blood up to the period
of her death.   The heart-sounds had become inaudible, and the
pulse imperceptible for several days.   The body had become
almost universally ecchymosed in broad irregular patches, sepa-
rated by portions of unaltered skin.   The trunk and thighs were
most discoloured.   The left side was clear, and yielded a loud
and harsh breath-sound, accompanied with râles, whilst the right
side, on which she principally lay, had become universally dull,
with loud muco-crepitus of a ringing and gurgling character.

No *post mortem* examination of the body was permitted.

This case has been reported at length, but with all possible
abbreviation, as a type of its class.   It exemplifies the usual
variation of symptoms having reference to the heart, and the
corresponding physical changes discoverable in the precordium.
There had been in the previous attack of rheumatism, endocar-
ditis and valvular lesion, involving mitral inadequacy.   The
murmur due to the lesion was, in the second attack, which was
accompanied with pericarditis, masked by the louder and more
superficial sound of pericardial friction.   On the temporary sus-
pension of the latter by liquid effusion, the mitral murmur again
declared itself; it was followed by the usual consequences of
mitral inadequacy, pulmonary congestion and hæmoptysis.   The
variations of friction-sound as to site, intensity, and rhythm,
dependent upon corresponding fluctuations in the amount of
liquid actually in the pericardium, are likewise well exemplified
in the case.

CASE II.—*Acute Rheumatic Pericarditis ; Recovery.*

Kate K., aged sixteen ; admitted September 24th, 1866, com-
plaining of pain in the hip and shoulder joints, and in the inter-
articular portions of the limbs generally, which commenced a
few days previously after exposure, whilst heated, to night air
Has had two similar attacks ; the first at the age of twelve years,
after a severe wetting, and the other two years later, from expo-
sure to cold.   When admitted she complained of intense pain in
the hips, shoulders, and knees, owing to which she found it

difficult to maintain the sitting posture even for a few seconds. Tongue moist and coated; copious acid perspiration; pulse full, and bounding; respiration 28, and interrupted; eyes brilliant, and right pupil dilated; great tenderness of the wrist joints, and general intolerance of pain; no cardiac symptoms, with the exception of sharp pain referred to the left mammary region Ordered, alkaline mixture, composed of acetate and bicarb. of potash; and, to appease thirst, which was very urgent, the "imperial" drink, consisting of a dilute solution of acid tartrate of potash, lemon juice, and sugar.

26th. Pulse 108, and full; complains of acute pain in the left arm, and likewise across the front of the chest during inspiration.

27th. Pulse 103, full, but occasionally irregular; bowels confined. A plaster to be applied to the region of the heart, composed of mercurial plaster (ʒss.) and powdered opium (gr. x.).

28th. Complains of pain over the heart; and, at the base, within a limited area about the diameter of a crown-piece, friction-sound is audible. Continue mixture.

29th. Precordial *frottement* audible over a larger area, but less loud than on yesterday.

30th. *Frottement* remarkably loud, and grating. Complains of pain at the heart, and want of sleep. Repeat plaster over heart, and let patient have gr. j. of watery extract of opium with gr. ss. of extract of colchicum every third hour, and alternately, and at equal intervals, half an ounce of lemon juice.

October 1st. Pains in the joints, for the last few days masked by the precordial distress, again constitute the chief cause of suffering. The affected parts to be rubbed with soap liniment, to which was added chloroform and tincture of aconite, each in the proportion of one-fourth.

2nd. Pulse 90, strong and regular; friction-sound very distinct.

3rd. Slept well last night; pulse 66; friction-sound of a much finer character; herpetic eruption appearing about the mouth and nose. Repeat pills.

4th Pulse 78, regular; slight pain behind xiphoid cartilage;

slept well last night. ℞. Sulphatis quiniæ, gr. xxiv; Acid. sulph. dil., ℈j ; Æther. chloric., ℨij ; Aquæ, ad ℥viij. St. ℥ss. 3tia q q. horâ.

8th. Pulse 120 ; *frottement* not audible, and cardiac sounds masked; increased extent of precordial dulness. Emplast. vesicat., region. cord. applicand. Haustus sodæ et potass. tartratis.

10th. Febrile excitement has subsided ; pulse 78 ; double friction-sound distinctly audible at base, and along the tract of the aorta. ℞. Hydrarg. perchlorid , gr. j ; Decoct. cinchonæ, ℥viij. St. coch. mag. ter in dies.

On the following day the quinine mixture was resumed, and in the course of the next, the patient was discharged convalescent.

CASE III.—*Acute Pericarditis supervening on Aortic and Mitral valve-disease ; Recovery.*

James W., aged seventeen, carpenter's apprentice, tall and delicate looking, was admitted to the Mater Misericordiæ Hospital, October 13th, 1866. One week previously he caught cold by exposure, and for two days subsequently had flying pains in the limbs, but no articular swelling. Two days later he was seized with acute pain in the region of the heart, which lasted till the day preceding that of his admittance, but had been, in some degree, mitigated by the use of sinapisms and warm applications. On the same day he began to suffer from dry cough, which was promptly brought on by assuming the recumbent posture. Patient is remarkably pallid and weak; pulse 132, and feeble ; respiration 42 ; cardiac impulse visible in the sixth intercostal space, in a vertical line with the nipple; it is tumultuous and attended with fremitus. At the apex an accompanying systolic bellows-murmur is audible, preceded by friction-sound ; and here the second sound cannot be heard. Over the entire basic region, but loudest at midsternum, and transmitted over front of chest generally quite to the clavicles, but not into the arteries of the neck, a double friction-sound is heard ; it is likewise heard in the left scapular region ; and in the infra-spinal fossa near the base of the scapula, a

double murmur of a cooing character is twice heard during each expiratory act, but ceases to be heard during inspiration ; and, on the anterior surface, a grating murmur, synchronous with the first sound, and quite superficial, is heard, with exceptional loudness during expiration, in the second intercostal space of the left side. Percussion and respiratory sounds normal over the entire chest ; tongue clean, and surface cool. Mercurial and opiate plaster to be applied over the heart. Perchloride of mercury, with compound tincture of cardamoms, to be given in infusion of calumba every third hour. To have gr. j of watery extract of opium at night, four ounces of wine, and good diet.

16th. Since last report the pulse has varied from 132 to 120 ; the latter being the rate to-day; it is now dicrotous: pupils small ; somnolence, thirst, and slight nausea, which latter may be due to the use of the expectorant mixture of the hospital for the purpose of alleviating his cough. Over the cardiac region generally, but most distinct at the apex, may be felt a well pronoun'ed fremitus ; at the apex, and likewise at the base, a double *frotte-ment* is audible ; but in the latter situation it is finer and *disin-tegrated* in character, by which it is at once distinguishable from double aortic murmur, likewise audible in the same situation, and from transmitted apex *frottement* In both situations friction-sound may be readily intensified by gentle pressure with the stethoscope ; it is audible beneath both clavicles by transmission.

24th. Patient's condition much worse ; total loss of rest and appetite ; respiration greatly embarrassed, and 42 in the minute ; pulse varying from 120 to 132, weak, dicrotous, and slightly visible. At extreme left apex is heard a loud creaking systolic sound ; here second sound is soft ; pulsation of apex tremulous over a space of about one and a-half square inch, a short distance below and to the left of the nipple, and here *frémissement* is perceptible. At base, especially towards the right. a double murmur is audible, which, from its compound character, viz, being, on the one hand, *superficial, grating*, and *interrupted ;* and, on the other, *soft, continuous*, and *synchronous* with the cardiac sounds, I judged to be due in part to pericarditis, and in part to disorganization of the aortic valves. It was audible likewise

beneath both clavicles, but more distinctly so on the right side. Carotids pulsating visibly. Three days since mercurial inunction was had recourse to for a period of two days, in the proportion of ℨij daily. Draughts containing hydrocyanic acid, and chloric ether (♏ij and ℨss respectively) were given to allay irritability of stomach, and gr. ȷ of opium at night to insure sleep.

26th. Is less restless; takes food well; at apex a soft systolic murmur is alone audible, but at base the compound double murmur may be heard; in left axilla a systolic murmur is faintly audible.

29th. There is physical evidence of congestion of the bases of both lungs, but this is more decided on the right side; patient passed a restless night; precordial dulness seems extended. To be dry cupped and poulticed over bases of both lungs.

November 1st. Pulse 126, and dicrotous; respiration 36; perspiration copious over head, neck, and chest; considerable increase in extent of precordial dulness, and less distinct impulse of heart; at apex a systolic murmur alone is audible, but it is likewise heard in left axilla; and at base a double murmur, but less loud than formerly. On yesterday (October 31st) patient complained of pain in right inferior lateral region, and here friction-sound was heard. Dry cupping and poultice to be applied at seat of pain.

6th. Has been sitting up for the last two days; slight laxity of bowels; mercurial to be stopped, and quinine to be substituted in gr. ij doses, ter in die, with ♏x of liquor of the pernitrate of iron.

7th. Feels and looks much better, and is taking more food; pulse 126, and of good volume; at apex of heart slight fremitus is perceived; and here a distinct double *frottement* is heard, masking the systolic murmur; double friction-sound is likewise audible at the base; but in the course of the aorta a double *bruit de soufflet* only; precordial dulness has become less in extent. The patient shortly afterwards left hospital convalescent.

The only remark I wish to make upon this case has reference to the modifying effect of respiration upon the friction-sound, as heard, at the date of the patient's admittance, in the second intercostal space, and at a corresponding point of the left back;

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the friction-sound, which was probably due to both posterior and anterior pericarditis, was rendered inaudible by the expansion, and consequent intervention of the lung during inspiration.

CASE IV.—*Acute Rheumatic Pericarditis ; Presystolic Friction-sound at Apex ; Recovery.*

James D, aged 23, a butcher ; admitted May 11th, 1869. On the 27th April he was attacked with pain in the knees, ankles, and wrists, but without swelling, save in the last named joints. On May 9th he experienced pain at lower end of sternum. When admitted he was pale and anxious looking ; perspired somewhat ; pulse 96 , urine loaded with lithates ; pressure over ensiform cartilage caused pain ; and here, as likewise at the base of the heart, a "to-and-fro" friction-sound was audible ; at the apex a single friction-sound was heard, which was presystolic in rhythm. ℞. Calomel. gr. ss ; Pulv. opii, gr. ss. M. To be given in form of pill every second hour ; poultice to precordium.

15th. Double friction audible throughout precordium, and transmitted to right second costal cartilage, but not into carotids ; pulse 72 ; respiration 36.

20th. Pulse 120 ; friction-sound loud, double, and heard universally over precordium. St. Hydrarg. perchlorid., gr. $\frac{1}{12}$, 4ta. q.q. horâ.

22nd. Friction scarcely audible ; precordial dulness much extended ; pulse 108. Ordered : Emplast. vesicat. precord. applicand. ; Mixt. alkalin. diuretic.

23rd. Pulse 96 ; friction scarcely audible. To have porter and wine.

June 2nd. Since last report this patient has had recurrent attacks of pain and swelling of a migratory character in the joints. The liquid effusion has been slowly removed from the pericardium, and the friction-sound has again become distinctly audible at the base. At the apex, no friction or other abnormal sound has been heard for the last week. Patient has been out of bed for several days ; he is now taking quinine, and recovering flesh. He was shortly afterwards discharged.

I will only remark upon the close resemblance, as to quality and rhythm, which the single friction-sound at the apex bore to the murmur of mitral constriction. This resemblance was further enhanced by the point (apex) at which alone it was audible. The distinction rested upon an occasional arythmic irregularity of the sound, the readiness with which it was intensified by pressure, its *superficial* character, and the coexistence of double *frottement* over the right ventricle and at the base

CASE V.—*Acute Rheumatic Pericarditis supervening upon Mitral and Aortic valve-disease; Recovery.*

James D., aged thirty-four, porter, of temperate habits; admitted into hospital September 29th, 1869. Had rheumatism sixteen years ago, when the joints were swollen. Four months ago began to feel uneasiness about the chest; and five days previous to admittance was again attacked with pain in the joints.

State on admittance Is anæmic; respiration normal; pulse 96, regular, and visible at the wrist; apex-beat one quarter of an inch inside nipple-line, and here a soft faint systolic murmur may be heard; second sound not intensified in pulmonary artery. At base a rough systolic, and a soft and faint postdiastolic murmur, were audible; both the latter were likewise heard in the course of the aorta, but the *former* only in the carotids. St. Citratis ferri, gr. v. 3tia. q.q. horâ.

About the middle of October, the man being then free from pain and fever, he was transferred to the care of my colleague, Dr. Cruise, in order to have a plastic operation done for hypospadias. Whilst under Dr. Cruise's charge he had a relapse of rheumatism, which was more acute than the previous attack, and was accompanied by pericarditis; a double but fine *frottement* was audible at the base of the heart; but, in order to educe it strong pressure with the stethoscope was necessary, and pain was then felt. St. Calomel., gr. j sing. horâ. Emplast. vesicat. precordio applicand.

In addition to the evidence of slight liquid effusion, afforded by the force of pressure required to bring out friction-sound, there was extension of the area of dulness, and faintness of the

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cardiac sounds Friction subsequently became again audible; on November 6th he was reported free from pain and fever, and at that date no *frottement* was anywhere to be heard. After a tedious convalescence he was discharged.

CASE VI.—*Acute Rheumatic Pericarditis and Myocarditis, with Pseudo-presystolic murmur; Mitral Reflux; Recovery.*

Lawrence O'B., aged twenty-four, paper manufacturer, temperate, admitted May 16th, 1868. On the second of May he was attacked with pain and malaise, and on the 9th was obliged to relinquish work Has now slight pain and swelling in feet, hands, knees, and shoulders; pulse 144, weak, and somewhat irregular; apex-beat in usual situation, but extending a little to left; slight dulness over bases of both lungs, and here respiration is feeble; no cough. To have "imperial" for drink, and a pill every third hour, consisting of gr. ss of extract of aconite, and gr iss of watery extract of opium; blister to right of midsternum where he complained of pain.

17th. Pulse 132, irregular, and full; perspires freely; sternal pain relieved by blister. At the apex of the heart a murmur is audible, presystolic in rhythm and rough in character, it was confined to the apex, left axilla, and back, in which latter situation it was very faintly audible.

19th. A distinct "to-and-fro" friction-sound audible at apex, where it is loudest, very superficial, and interrupted; it is likewise heard at the base, but less distinctly; no fremitus or increase of precordial dulness; pulse weak, failing, and irregular at wrist, where it cannot be counted; but the pulsations of the heart are 168 in the minute; respiration slightly hurried, but not embarrassed; no head symptoms; is pale; perspired copiously about head and chest last night, but feels much improved to-day. To have blistered surface dressed with mercurial ointment, and to continue pills

June 10th. Owing to temporary illness which compelled me to relinquish hospital duty, I lost sight of the patient, having placed him under the care of my colleague Dr Hughes To-day I found that in the interim he had had double pleuritis with effu-

sion; right side still dull to a level slightly above nipple, and left side to a less extent; pulse rapid and feeble, but regular; heart acting at rate of 160 in the minute, and at the apex an ill defined "rub" precedes the first sound; elsewhere both sounds are sharp and clear; precordial dulness normal in extent; he is remarkably pale; and has been for some time taking iron under the directions of Dr. Hughes.

16th. Pulse steady, and 132; "rub" preceding first sound of heart has assumed still more the character of the murmur of mitral constriction. The patient has been out of bed during the last two days.

23rd. Pleuritic friction-sound on both sides; pulse 132, regular; is recovering colour; presystolic friction-sound at apex is very faint. Ordered: Infusi senegæ c. carb. ammoniæ.

30th. A faint pleuritic friction-sound is heard in the left lateral region; and preceding the first sound of the heart, at the apex only, a rough jarring murmur, which extends quite up to the first sound; impulse strong and extended, and presystolic *frottement* audible to right as far as middle line, but with decreasing distinctness. Discharged, and went to the country for change of air.

July 20th. Visited the hospital to-day; feels much improved; pulse regular, 120, and of moderate volume; apex-beat in nipple-line, and here, as also at left base, a single friction-sound of presystolic rhythm is audible. To have iron and chloric ether (of each ℳ lxxx), in ℨviij of infusion of quassia, to be taken in ℨss doses thrice daily; also cod-liver oil (ℨss) with syrup of iodide of iron (ℨj), thrice daily after food. May resume light work, and take tepid baths. Discharged.

March 8th, 1870. Readmitted to-day; is now generally anasarcous; swelling commenced in lower limbs and in face a month previously, accompanied by epistaxis and dyspnœa; a fortnight later swelling of genitals set in, as likewise cough and expectoration. Is now pale; face, lower extremities, and genitals much swollen; cough, and universally diffused râles; pulse regular, 96; apex-beat in nipple-line, and beneath sixth rib; impulse is diffused, a loud systolic bellows-murmur is audible at the apex; no intensification of second sound; urine contains

a good deal of blood, likewise albumen, and tube casts denuded of epithelium but presenting a few granular epithelial particles.

After active purgation with podophylline, hot and dry air-baths were given, with the effect of rapidly dissipating the dropsical effusion; the kidneys at the same time began to act vigorously, excreting about two quarts of urine in the course of the night, and of a more natural colour. In the course of a few weeks he was discharged much improved in general health, and quite free from dropsy.

The last attack was manifestly one of acute renal anasarca, the heart continuing to exhibit the traces of former endo-pericarditis. The pseudo-murmur at the apex might very readily have been mistaken for presystolic mitral murmur; but in this, as in all similar cases, the differential diagnosis was not only possible, but not difficult, quite irrespectively of all previous knowledge of the case, attention being given to the superficial localization and the *unsteadiness* of rhythm of the sound.

CASE VII.—*Acute Rheumatic Pericarditis ; Mitral Regurgitation of non-organic origin ; Recovery.*

Mary N., aged thirty, domestic servant, admitted December 8th, 1870, suffering from acute rheumatism engaging several joints of the lower limbs. Two days previously she complained for the first time of pain in the shoulders, arms, and hands. These joints became swollen; but in the course of the two succeeding days the disease shifted to the joints of the lower limbs, which, at the date of admittance, were swollen and tender. Perspiration copious; pulse 120, and full; tongue loaded and moist, face remarkably pallid. A faint thrill was perceptible with expiration at midsternum; and here, but not elsewhere, a sharp and single friction-sound, postsystolic in rhythm, was audible; action of heart abrupt. Alkaline mixture, and mercurial and opiate plaster to precordium.

13th. Friction-sound, double systolic and fine.

14th. Double friction-sound, viz., systolic and postdiastolic respectively in rhythm, coarser in character, and audible at base exclusively; pulse soft, regular, and 108; no carotid pulsation,

dyspnœa, or epigastric tenderness; slight increase in extent of precordial dulness, which extends vertically to one and a-half inch above level of nipple, and horizontally to right edge of sternum.

17th. Friction-sound of last mentioned rhythm coarse and audible at ensiform cartilage; precordial dulness less extensive; at apex first sound is rather prolonged; no carotid pulsation or epigastric tenderness. Has been taking for the last week a sleeping draught at night, composed of twenty to twenty-five minims of Battley's sedative, and four ounces of wine daily.

Two days since pain and swelling returned to wrists, right ankle being similarly affected.

25th. Dulness still extended somewhat beyond the limits of the superficial precordial region. Since date of last report pulse has varied from 108 to 120, but was full and regular; no precordial pain; respiration slightly quickened. On yesterday patient had a slight attack of dysentery, which still continues. A soft but indubitable systolic murmur is now audible at left apex. Alkaline mixture stopped, and an astringent ordered, consisting of aromatic chalk powder, tincture of catechu, and compound tincture of cardamoms.

27th. Dysentery still troublesome, chiefly in the morning; pulse 120; tongue dry and clean; breathing and pericardial dulness as at date of last report; at base of heart, and less distinctly at lower end of sternum, a sharp friction-sound is heard immediately succeeding the first sound; no other friction-sound audible, but at apex a distinct bellows-murmur exists, and extends into axilla. Emplast. vesicat. precordio applicand.

January 20th, 1871. First sound at base double; second sound sharp and clicking; no murmur audible anywhere; no systolic dimpling of surface, and no *frémissement*.

The *single* character of the friction-sound at the onset, and likewise at the close of pericarditis, the rhythm having been double in the interval, is noteworthy in this case. The development of a systolic murmur at the apex in the course of rheumatic pericarditis, might have been regarded as conclusive evidence of endocarditic complication; and, indeed, for some time I regarded it in that light. The subsequent history of the

case, however, supplies, in my opinion, a complete refutation of that view. A month subsequently, after a course of tonic and chalybeate medicines, the condition of the patient having in the interim undergone a decided general improvement, the murmur at the apex no longer existed. Where valvular inadequacy has once existed as a consequence of endocarditis, I cannot admit so complete a restoration of the valve to its pristine condition, as would preclude the continuance of reflux in some, however small, a degree. On the other hand, it seems not unwarrantable to conclude that, in a condition of anæmia and general debility, such as that witnessed in the course of slow convalescence of this patient, a state of atonicity or weakening of the muscular walls of the left ventricle of the heart would have occurred, in which, at the acme of systole, a yielding of some portion of the ventricular wall, under the centrifugal or counter-pressure of the contained blood, might have taken place, by which a valve-segment might have been retracted from the orifice through the medium of one of the papillary muscles and the corresponding tendinous chord, and regurgitation as a consequence have taken place. Improvement in the general condition of the patient under tonic and restorative treatment would, in that view, afford an adequate explanation of the cessation of murmur. It is note-worthy, that in the progress of recovery and cessation of this murmur, reduplication of the first sound at the apex was observed, constituting, no doubt, a stage of redux transition from systolic murmur to a normal first sound.

CASE VIII.—*Hypertrophy and Dilatation of Right Ventricle; Partial Hypertrophy of Left Ventricle; Chronic Non-rheumatic Pericarditis; Death.*

John S., aged about forty-five, van driver, admitted January 17th, 1871. Has been short-breathed since childhood, and spat some blood a few months ago. Two months prior to admittance feet and legs began to swell.

When admitted he was suffering from aggravated dyspnœa, being unable to lie down; the face livid, and the cervical veins distended to the utmost degree, but not pulsatile. The lower

limbs generally were œdematous, tense, and livid; the genitals also swollen; radial pulse slow, soft, large, and regular. There was physical evidence of aggravated pulmonary emphysema and bronchitis, with congestion and œdema of the bases of both lungs.

Cardiac impulse was felt at the ensiform cartilage, where both sounds of the heart were distinctly audible, the first being rather sharp and clear. There was likewise audible in this situation an equivocal murmur of a harsh and somewhat grating quality, accompanying the first, and also, but very faintly, the second sound. The cardiac sounds were elsewhere less distinctly audible, and free from murmur. The urine was of normal specific gravity, and contained a trace of albumen. ℞. Tincturæ digitalis, Spirit. æther. nitr., aa. ℥x, in camphor water, every third hour; counter-irritation to chest, and a moderate allowance of whiskey punch. On the following day the pulse was irregular, but otherwise there was slight improvement. On the 19th he was incoherent; became delirious; and died comatose on the morning of the 23rd.

Post mortem examination of body. About thirty ounces of serum were found in each pleural cavity; the lungs were generally emphysematous, and congested at the base, where a few nodules of sanguineous extravasation were found. About three ounces of serum, but no fibrinous flakes, were found in the pericardium. There was a large "milk spot," two inches in diameter, on the anterior surface of the right ventricle, and on its apex a rough patch of exuded lymph; the free surface of the pericardium, to the extent of about one and a-half inch in diameter, was rough and jagged; the serous investment of the right auricle and appendix generally was likewise rough and opaque, and that of the left auricle also, but to a less extent. The heart weighed twenty and a-half ounces; it was globular in figure, and was formed in greatest part of the right ventricle, of which the apex was entirely composed. The right chambers contained some partially decolorized fibrin, which quite filled the auricular appendix and overlay the orifice of the pulmonary artery. Both these chambers, in the remaining portion, were full of dark coagulated blood. The right auricle was dilated, and the right ventricle greatly dilated and hypertrophied; its wall, midway

between the base and the apex, was three-eighths of an inch thick. The tricuspid opening freely admitted the four fingers and thumb to the roots. Both the tricuspid and right semilunar valves were normal; the left auricle was unaltered; the left ventricle somewhat hypertrophied; mitral and aortic valves normal and competent.

Several days anterior to death, the diagnosis was made of pericarditis, cardiac hypertrophy and dilatation, pulmonary emphysema, bronchitis and œdema, and absence of valvular disease.

In a state of extreme engorgement of the right chambers there is no murmur, even though there be tricuspid reflux, owing to permanent distension and enfeeblement (*asystolia*) of the right ventricle. It is in cases of tricuspid valve-disease, where weakening and distension of the right chambers do not exist, that murmur at the tricuspid orifice is developed.

CASE IX.—*Mitral Regurgitation, and Hypertrophy, of some years' standing; Recent Pericarditis and Pneumonia; Death.*

William H., aged eighteen, shop-assistant, admitted March 17th, 1871. Eight days previously, after a walk, he was seized with severe pain across the front of the chest and cough accompanied with blood-stained sputum. Has never had rheumatism, but was regarded as a delicate person When admitted he was in a state of collapse, the pulse being entirely suppressed in the right radial, and very feeble and quick in the left radial artery; surface cold; cheeks, lips, tip of nose, external ears, and finger-ends were quite livid; respiration quick, shallow, and painful. Precordial dulness was not perceptibly increased in extent; cardiac impulse strong and double; the second sound was double, the first element accompanying the second or diastolic impulse, and everywhere loudest; apex-beat in nipple-line and fifth intercostal space, and here a blowing, substitutive, systolic murmur was audible, and strictly limited to area of apex. At the date of admittance this murmur was not audible, but was detected on the following day; on the second day it was again inaudible; but on the third day (20th), and subse-

quently, up to the close of life, it was distinctly heard. Hepatic dulness extended four inches below costal cartilages, and there was tenderness at the epigastrium. The lower and posterior portion of the right side was dull, and bronchial respiration and crepitus were audible over the posterior right base. Sputa viscid and amber-coloured. ℞. Liquor. Hoffman.; Spirit. æther. nitr., aa. ℥ss; Tincturæ digital., ℳv, cum aqua camphoræ, ut ft. haustus. 3tia, qq. horâ sumend. Whiskey and water frequently in small quantities, and dry cup and poultice right side at seat of dulness.

18th. Pulse stronger; face now of a crimson hue; moans with pain referred to a point one inch below and to inner side of nipple; double impulse very distinct; one (the stronger) accompanying the first sound of the heart, the carotid pulse, and the apex-murmur; the other, less strong, but decided, and everywhere synchronous with the first element of the double second sound. Cupping to four ounces over right base of right lung, and two leeches at site of pain in precordium, to be followed by a warm poultice. ℞. Calomel., gr. ss; Pulv. Jacobi, gr. j. M. Ft. pulv. quinta. Una qq. horâ sumend.

21st. Has now taken twenty-two powders, and exhibits the early signs of mercurial action; is quite relieved from pain; respiration easy; pulse on left side 108, and strong, but imperceptible on right side. ℞. Sulph. quiniæ, gr. ij; Sulph. morphiæ, gr. ₁⁄₁₆. M. Ft. haustus. 3tia qq. horâ sumend. Poultice to side to be continued.

22nd. Slept well, and is much improved. To have a chop.

April 3rd. A few days since, the base of the left lung was attacked with pneumonia, for which he was cupped and blistered. The sputa were for some days deeply tinged with florid blood; respiration much embarrassed, and the pulse on two several days registered 192. This morning there is sharp diarrhœa; respiration 40. The left pleural cavity was found nearly full of liquid, dulness extending within a short distance of the clavicle; the upper portion, however, to the extent of a hand's breadth vertically, both before and behind, yielded muffled tympanitic resonance. Respiratory sound was nearly suppressed, but not altered in character over the entire left side; ægophonic voice was

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audible beneath the spine of the scapula; and vocal thrill was entirely abolished on the left, whilst it was distinctly perceptible on the right side. The heart was displaced to the right of the sternum, and pulsated in the right fourth intercostal space, half an inch inside the nipple-line. The first sound was free from murmur; the second faintly double, its first element being accompanied as before by diastolic impulse. Puerile respiration is audible over the entire right side, which is clear on percussion to middle line of sternum, beyond which absolute dulness exists, modified superiorly by muffled tympany as already stated.

℞. Tinct. digitalis, ʒj; Spirit. æther. nitrosi, ʒiv; Sp. juniper., ʒij; Decocti scoparii co., ad ℥xij. St. ℥j 3tiis horis. Whiskey in small doses, and frequently.

April 8th. Died suddenly last night, with symptoms of syncope.

Post mortem on the 9th. About eight ounces of serum in sac of pericardium, which was not attached to heart, or lined by false membrane, but thick, opaque, and perfectly smooth throughout. Heart somewhat enlarged, weight eleven and a-half ounces; left ventricle hypertrophied and dilated; it was half an inch thick except at apex, which was very thin; structure of heart and valves absolutely healthy. A yellow fibrinous thrombus extended from the right auricular appendix into the right ventricle, but without blocking the tricuspid orifice, or entangling the valve. The exterior of the pericardium on the left side was thickly covered with miliary tubercle of the size of duck-shot; it was blood-stained, and firmly attached to the inner surface of the left lung, which presented a similar appearance. The left lung was covered with false membrane, reduced in volume, and carnified, with the exception of the apex, which was attached to the chest-wall, and contained some air; in the base of this lobe there was a nodule of sanguinous extravasation, and in its centre a caseous mass as large as a hazel-nut; a few caseous masses of smaller size existed in the apices of both lungs. The lower lobe was nowhere adherent, and lay across the spinal column, its inner surface being attached, as already stated, to the pericardium, which lay to the right of the middle line; the base of the heart corresponding to the right fourth intercostal space in

the nipple-line, and the apex on the same level, but internally to the base. The areolar tissue of the anterior mediastinum was of a gelatinous character, and infiltrated with serum. The right lung was partially attached by its anterior surface to the chest-wall; the base was dark and congested, but resonant. Several large lymphatic glands of stony hardness surrounded and com-pressed the right subclavian artery; the bronchial glands were likewise enlarged. Several pints of dark blood-stained serum were found in the left pleura, displacing the pericardium to the right side.

In relation to double cardiac impulse, I have already ex-pressed my opinion, that the second shock is due to the abrupt expansion or diastole of a dilated and hypertrophied left ven-tricle. In the case above narrated, the first and stronger im-pulse coincided with carotid pulsation, and preceded, by a very slight but appreciable interval, that of the radial artery; it was, therefore, the representative of the normal impulse of the heart; the second and weaker impulse was no less certainly ventricular, because felt in the same situation as the first, viz., at and immediately above the apex only; and diastolic, because coincident in time with the first and louder element of the double second sound, which I have already shown to represent the nor-mal second sound In reference to the doctrine of Hope, imply-ing a necessary dependence of double shock upon adhesion of the pericardium, as the starting point of the pathological series issuing in hypertrophy of the heart, and as illustrating the error involved in that doctrine, the case is likewise interesting No adhesion whatever existed, and yet double impulse was well pronounced, and associated with dilated hypertrophy of the left ventricle exclusively.

Temporary and fluctuating mitral reflux murmur I can attri-bute only to partial yielding of the wall of the left ventricle in systole, and consequent displacement of the valve. It might be supposed due to localized friction on the left side and exterior of the pericardium, roughened by tubercular deposition and recent exudation of lymph; but in that case its rhythm would have been in some degree respiratory, its quality in all probability harsh, its regularity much deranged, and its seat of greatest in-

tensity more remote from the mesian line. Yet, I am willing to confess the existence of some doubt in my mind that it was not caused by exo-pericardial friction, because of the actual firmness of the ventricular walls as determined by the autopsy, and the final cessation of the murmur on the third day after admittance.

Localized tubercular deposition on the exterior of the pericardium and corresponding portion of the pleura, is not common, and is worthy of being recorded as the possible cause of an acoustic phenomenon liable to be confounded with endocardial murmur. Suppression of either radial pulse, whilst the other is perceptible, will be found due, in ninety-nine out of one hundred cases, to the pressure of an aneurism, or of enlarged lymphatic glands in the neck or axilla. If, therefore, aneurism be excluded, the diagnosis of glandular pressure upon the main artery of the limb may, with equal confidence, be made.

CASE X.—*Circumscribed Pericarditis confined to the Root of the Pulmonary Artery.*

—— Y., a girl of sandy complexion, aged about twenty, admitted in May, 1871. Has had rheumatism, and at the date of admittance suffered from that disease in a sub-acute form, the attack having commenced three weeks previously. The left hand and forearm were stiff and wasted, and there was considerable tumefaction, without tenderness, over the left internal condyle of the humerus. There was slight pain and swelling of the joints of the right arm; scarcely any febrile action; pulsation and sounds of heart normal, with the exception that, over the inner portion of the left third intercostal space close to the sternum, a harsh grating sound was heard accompanying the first sound of the heart; it was strictly limited to the situation mentioned, audible over a space not larger than the bell-end of the stethoscope, intensified by pressure, and barely audible when pressure was not made. There was no murmur at the right base, or in the neck.

After a residence in hospital of about a month's duration, she left on the 1st of June, improved in general health, and free

from pain, but without any alteration having taken place in the character or intensity of the murmur.

In addition to "auricular pericardial friction,"[*] characterized by *frottement* of presystolic rhythm, and limited to one or both auricles, basic pericarditis, without implicating the ventricles proper, may likewise yield an attrition-sound simulating endo-cardial murmur. In such case the friction-sound is strictly sys-tolic in rhythm, and due to pericarditis involving the roots of the great arteries exclusively. It is remarkably persistent.

The case just narrated was an example of this kind; the effused fibrin having been limited to the root of the pulmonary artery.

CASE XI.—*Pericarditis with Effusion ; Recovery.*

Margaret B., aged fifty, admitted October 8th, 1867 ; health good up to the preceding summer, when, after the sudden retro-cession of a cutaneous eruption (kind not known) she began to suffer from palpitation. A month previous to admittance she was attacked with pain in the left side, accompanied with diffi-culty of breathing ; swelling of feet and abdomen subsequently took place.

At the date of admittance the feet and legs were much swollen and slightly congested ; there was some liquid in the peritoneum, and a good deal of subcutaneous serum ; pulse scarcely to be felt at wrist, but counted by heart it registered 138 ; cardiac action feeble and irregular, and no impulse perceptible ; sounds masked ; precordial dulness extended to the left one and a-half inch out-side nipple-line, to the right edge of the sternum, and upwards one and a-half inch above level of nipple ; respiration embar-rassed , cough, with mucous expectoration. St. Calomel , gr. ss, secunda q. horâ ; blister to precordium, and chloric ether (ʒss) in camphor water every third hour ; wine. Dorsum of left foot punctured to allow escape of serum.

14th. Pulse (counted by heart) 72, very weak, and impercep-tible at wrist ; precordial dulness less extensive towards the left, and extending on right side only to middle line ; cardiac impulse now perceptible ; both sounds distinctly heard, and a rough,

* Hyde Salter, *Lancet*, July 29th, 1871

jarring, and superficial quasi-murmur with the first sound is occasionally heard over the base, loudest to the left of the middle line, and faintly audible at the apex.

15th. ℞. Emplast. hydrarg., ℨss; Pulv. opii, gr. x. Ft. emplastr., super cord. applicand. ; stop pills, and continue mixture ; puncture on dorsum of left foot discharging serum freely. Returned home, at her own desire, the pulse then being intermittent and irregular, and the cardiac phenomena as previously described.

CASE XII.—*Chronic Pericarditis and Pleuritis ; Suppression of Right Radial Pulse ; Death.*

James F., aged thirty-eight, bricklayer, admitted February 10th, 1871. Has had occasional hæmoptysis during the last six years ; breathing has been short the last two years ; has drunk hard. On the 1st February began to experience a sensation of fulness and dragging in the lower part of left side ; this continued up to the 7th inst., when he was suddenly attacked with dyspnœa of an urgent kind. The paroxysm soon subsided, but his breathing continued much oppressed to date of admittance. Breathing was then greatly embarrassed, thirty-six in the minute ; thoracic movement free and equal on both sides, but supra-sternal and left supra-clavicular space sank in with respiration. Resonance was normal on both sides ; and on right side respiratory sound was loud and harsh, with sparse râles, but on left it was everywhere feeble, and in infraclavicular region almost *nil*. Pulse 120, full and regular on left, but on right side it was scarcely perceptible ; the artery was tortuous, and the fingers clubbed.

March 5th. There is now general bronchitis, with *gargouillement* in left front and in right posterior base. Friction-sound has existed for some weeks past over lower left side, and to-day it is audible over left boundary of pericardium, where it coincides with both respiration and cardiac impulse, but only in the recumbent posture. For some weeks before death the left side was absolutely dull, and respiration was faintly audible only behind. Died, April 26th. About four pints of blood-stained

serum were found in the left pleura; the left lung was enveloped throughout in a thick layer of false membrane, and was adherent to the pericardium, but nowhere to the chest-wall. The lung was reduced in volume and partially carnified, and in its apex were some masses of crude tubercle, and a smooth-walled cavity the size of a filbert. With this cavity no bronchial communication could be traced, nor did water injected into the principal bronchus find entrance into it. The right lung was universally adherent; the pericardium greatly thickened and rough on its inner surface; it was incorporated with both pleuræ by false membrane half an inch thick. In the pericardial sac was a pint of amber-coloured serum. The surface of the heart and the roots of the great vessels were completely enveloped in false membrane of a reticular and granular character, and two to three lines thick. The heart, which was pointed at the apex, weighed fourteen and a-half ounces; the outer surface of the right ventricle, to the depth of one half its thickness, was composed of pure adeps; its muscular structure was pale and everywhere granular, and no striæ were visible. The left ventricle was half an inch thick, and dilated; the aorta was slightly atheromatous in a few spots; valves all healthy; some flakes of fibrin were found in the right ventricle, extending into the pulmonary artery In the recumbent posture the left lung advanced as far as the investing false membrane allowed, and by approximating the pericardium to the heart, became accessory to the friction-sound which accompanied both the movements of the heart and of the lungs. The extra weight of the heart was in greatest part due to the thick layer of false membrane by which it was enveloped; and the great respiratory distress exhibited during the last weeks of life was in a great degree due to fatty transformation and failure of the right ventricle.

I regret that no examination of the right supraclavicular region was made, with a view to ascertaining the cause of suppression of the right radial pulse.

CASE XIII.—*Rheumatic Endo-pericarditis; Recovery.*

Jane A., aged twelve years, admitted October 31st, 1870, for subacute rheumatism; ill a week before admittance. When she

was first examined a suspicious prolongation of first sound was detected at the apex, which, after a few days, assumed all the characters of a murmur.

November 20th.   Apex-murmur, distinct, systolic, and soft; double pericardial friction-sound over precordium generally, extending upwards to left clavicle, but not heard at apex, the second element being postdiastolic; pulse 108; no pain in joints, or over heart.

25th.   Both elements of friction-sound synchronous with sounds of heart.

29th.   One friction-sound only audible at base, systolic in rhythm, and harsh in quality, but *not* audible at right second costo-sternal joint, nor in the carotids; no fremitus.   Is convalescent, and taking iron and quinine.

30th.   No basic murmur whatever audible, but at left apex a distinct soft systolic murmur exists.   Discharged.

CASE XIV.*—*Rheumatic Pericarditis engaging especially the Root of the Pulmonary Artery; Tubercular Softening of Left Lung; Recovery.*

Peter D , aged twenty-two years, carriage-smith; had rheumatism two years anterior to date of admittance on the 15th of February, 1869.   Taken ill eight days previously with pain in knees and general muscular tenderness.   When admitted, his wrists were swollen and red; perspired freely; heart's action obscure , sounds muffled and of a suspicious character.   St. Lemon-juice, ℥ss, 3tia q q horâ.

20th.   Pulse 132, full and dicrotic, but soft; respiration 72 Last night he was attacked with pain "in the stomach," which, doubtless, was precordial, and was relieved promptly by the application of two leeches by the resident pupil, Mr Kelly   Precordial dulness now extended, and sounds scarcely audible at apex   At left base a faint friction-sound exists.   To have calomel, gr ss, with James' powder, gr j, every hour; and

drachm-doses of spirit of nitrous ether and spirit of juniper, every second hour.

21st. Diarrhœa; pulse 132; respiration 48; extended precordial dulness, and no friction-sound audible; bronchitis, and congestion of posterior bases of both lungs. Mercury to be stopped, and patient to be dry cupped behind. To have thrice daily, in mixture, gr. iij of compound powder of chalk, with ℳ v of tincture of opium.

23rd. Pulse 132; respiration 60; heart-sounds obscure and weak. To have gr iij of citrate of iron and quinine thrice daily; wine and porter.

26th. Pulse 108; respiration 48; over left third intercostal space near the margin of the sternum, i.e., over root of pulmonary artery, there is a "to-and-fro" friction-sound, each element being double, and counting 156, whilst the radial pulse was only 108; one inch lower down, at level of nipple, only a single rub is heard, which is synchronous with the radial pulse.

Friction was suspended on the 27th, but was again heard on the 28th, and on the 29th it was masked by loud râles. On that day the patient was very weak and nervous; breathing greatly embarrassed; both bases dull posteriorly. A large blister was applied here.

March 3rd. Pulse 120; respiration 60; cough troublesome, and expectoration thin, frothy, and slightly tinged with blood; both sides comparatively dull behind, with a peculiar tympanitic note below angle of left scapula, over a space of a hand's breadth.

4th. Pulse 108, soft, full, and compressible; respiration 36; less cough; loud gurgling râles in upper left front.

8th. General softening of left lung; apex beat in fifth intercostal space, half an inch outside nipple-line; pulse 96; slight hectic flush, and perspiration at night; expectoration frothy and mixed with blood.

11th. Pulse 96; no perspiration; less cough; respiration 24; râles and dulness above nipple diminished; apex-pulsation in nipple-line and fourth intercostal space; small purpuric spots on feet and legs; has been out of bed for the last two days.

April 8th. Slight cough, with night sweats ; breathing quick ; tongue clean, and appetite good ; has gained flesh.

In reference to the subject under discussion, the most noteworthy feature in this case is the multiplication of friction-sounds in the site of the pulmonary artery, a consequence, no doubt, of the undulatory movements of that vessel in alternate distension and reaction ; and this, whilst one inch lower down a single *frottement* only existed, which was synchronous with the first sound of the heart.

CASE XV.—*Renal Disease ; Chronic Pericarditis ; Cardiac Hypertrophy and Hepatic Congestion ; Death.*

John C., aged twenty-eight years, brewer's carter, of sandy complexion, admitted September 19th, 1870. Fourteen weeks previously his feet became swollen, and a fortnight later he had palpitation, and he noticed enlargement of the liver. Three days before admittance he had bilious vomiting and coughed up some florid blood, and on the day of admittance he had a rigor Never had rheumatism. When admitted he was pale, and unable to retain food or drink of any kind. The vomited matter was tinged with bile. Pulse 84, regular, and very weak ; feet swollen ; urine 1·02, containing lithates, but no albumen ; hepatic dulness extended down nearly to the umbilicus, and upwards to the level of the fifth rib ; epigastric hollow obliterated, and hepatic surface smooth, but very tender to pressure ; no discoverable splenic enlargement ; urgent dry cough, by which pain in region of liver is aggravated. The apex of heart pulsated two inches outside the nipple-line, in the sixth intercostal space ; and here, for a period of two days, a *bruit de cuir neuf* was heard, which on the first day was systolic, and on the second day diastolic in rhythm. Small doses of calomel and James' powder were given, but promptly rejected ; leeches and poultices were applied at the epigastrium ; wine, beef-tea, and ice were administered. Much relief was experienced from the leeching.

For a period of about ten days the friction-sound now ceased to be audible. On October 2nd it was again heard, and was

then systolic in rhythm; and on the following day, whilst the apex *frottement* preserved the same rhythm, a double friction-sound of a decided character was audible over the remainder of the precordium.

The general condition of the patient was now improved; pulse regular, stomach less irritable, cough nearly gone, and a few râles only audible. A mercurial and opiate plaster was applied to the precordium.

5th. A fine systolic *frottement* exists at apex, but is not audible in axilla; elsewhere in precordium a rough double friction-sound is heard; œdema of feet, legs, and scrotum.

16th. Since date of last report the prepuce has been repeatedly punctured to facilitate micturition; the pulse varied much, occasionally intermitting; the stomach continued irritable, and the precordial sounds varied little. On this day, however, cardiac action and sounds being normal, no attrition or other sounds are anywhere audible; precordial dulness of normal extent; and stomach capable of retaining solid food.

18th. The phenomena of cardiac action are remarkable; both sounds are seemingly reduplicated, so that four sounds, occurring in rapid succession, correspond to a single beat of the radial pulse, which is regular, of moderate strength, and eighty-four in the minute. These reduplicated sounds are in some degree masked, but they mimic the normal sounds of the heart, and are audible throughout the precordium. (*Bruit de rappel.*)

On the following day (19th), only three sounds were audible, the first two of which corresponded in rhythm with the cardiac impulse, and represented the normal first sound; and on the 20th, the sounds were again only two in number, and of the character exhibited before the 18th, *i.e.*, slightly dulled but otherwise normal. The patient had a sensation of "dragging" at the apex with each action of the heart. Digitalis was now given in ♏x doses of the tincture every third hour, with an equal quantity of spirit of nitrious ether. Urine increased in quantity, and œdema of genitals and of lower limbs decreased; cough had nearly ceased, but some bloody sputa had appeared.

26th. Great œdema of left arm and hand, and of lower limbs; pulse 108 and weak, but regular; congestion of posterior base of

right lung; sputa bloody, and patient somnolent, but hopeful. A loud *bruit de cuir neuf* is audible all over precordium, also beneath both clavicles, on both sides of back, and along spine to lower dorsal region; its rhythm is double, one portion being synchronous with the impulse and first sound, and the other posterior in time to the second sound, *i.e.*, one was systolic, and the other postdiastolic. *Frémissement* perceptible all over precordium, and a tremulous movement of the intercostal spaces to some extent visible, corresponding in time with the double *frottement*. There is no retraction of the epigastrium or intercostal spaces, and no distention of the jugulars, or venous collapse in the neck.

27th. The patient complains of acute pain in the region of the heart; at the base a loud harsh double *frottement* is audible, and at the apex a double attrition-sound of similar rhythm, but higher pitch, and musical quality. Precordial pain relieved by a leech, from the bite of which there was rather copious bleeding, and on the following day the patient was very weak ; pulse 96, regular, and feeble ; cardiac action likewise feeble, and friction-sounds less loud but otherwise unaltered, and still faintly audible under both clavicles. Œdema of genitals and lower limbs now extreme, and seems likely to give rise, by tension, to gangrenous inflammation and sloughing of the integument of the thighs.

28th. Punctures freely made, from which there was a copious flow of serum. The haggard and wasted aspect of the face now characteristic ; breathing much oppressed, and stomach occasionally rejects food and drink. Death occurred Sunday, October 30th.

Post mortem examination twenty-four hours later. There was thickening and opacity of the peritoneum, but no ascites. The capsule of the liver was thickened and white over a space of one and a-half inch in diameter at each side of the suspensory ligament. The liver was somewhat enlarged, but otherwise unaltered. Each pleural cavity contained about a quart of clear serum. The lungs were sound, with the single exception that the lower lobe of the right was congested and œdematous. In the pericardium about six ounces of viscid amber-tinted liquid were found, which,

on microscopic examination, exhibited blood-corpuscles and compound granular cells. The heart was globular in figure, and hypertrophied; it weighed eighteen ounces. The left ventricle was much hypertrophied, and somewhat dilated, its walls below the base being more than half an inch thick. The right chambers were, as to capacity and thickness of walls, strictly normal; valves everywhere normal and competent. On the anterior surface of the right ventricle a patch of rough fibrinous deposit was found : it was three-fourths by half an inch in diameter, and coarsely striated at an acute angle with the long axis of the heart ; and on the parietal layer of the pericardium a similar patch, corresponding in situation and extent, existed. On the posterior surface of the heart two patches of the same character were found ; one upon the visceral, and the other on the parietal pericardium. A large patch was likewise found on the right auricle ; and another, but smaller, on the left apex. In these several situations flakes of white and partially attached fibrin depended from the surface of the heart. The fibrin was everywhere white and skein-like, and entirely devoid of blood-vessels or spots ; it loosely adhered to the heart, and, microscopically examined, was filamentous in structure Kidneys small, granular, and pale ; on section, cortex was thick, and presented excess of fibrous structure, with much fat in granules and large masses ; surface of section was generally pale, and pyramids reduced in size.

This case well illustrates the unsteadiness of the abnormal sounds, as to number, rhythm, and site. The loudness and the extensive diffusion of the *frottement* in connexion with hypertrophy of the heart ; and the blanched appearance, low organization, and non-adhesive character of the effused lymph, distinctive of chronic pericarditis of renal origin, are likewise noteworthy.

CASE XVI.—*Circumscribed Pericarditis engaging the Root of the Pulmonary Artery only ; Persistent Frottement ; Recovery.*

Mary A. K., aged thirty-two years, admitted into hospital, January 13th, 1872, for mild rheumatism engaging the smaller joints of feet and hands, and attended with perspiration and vascular excitement. Had a similar attack twelve years pre-

viously.   Two days after admittance a faint double friction-
sound was heard over the root of the pulmonary artery; it was
not attended with *frémissement*, and was inaudible elsewhere.
The patient was restored to perfect health in the course of a
fortnight, under alkaline treatment; but the friction-sound in
the original situation, to the left of the sternum, continued un-
altered at the date of her discharge.

CASE XVII.—*Circumscribed Pericarditis; Recovery.*

Kate M., aged fourteen years, admitted February 7th, 1872
Two days before, she complained of pain in the feet, and on the
date of admittance, in the right hand, the fingers of which were
swollen.   Pulse 96, regular; pericardial systolic *frottement* con-
fined to root of pulmonary artery, and accompanied with feeble
fremitus at acme of expiration.   Discharged cured after a few
weeks' residence, the pericardial attrition-sound having con-
tinued unaltered up to that date.

CASE XVIII.—*Rheumatic Pericarditis with Effusion; Recovery*

James F., aged seventeen years, admitted under Dr. Nixon,
November 17th, 1871.   At Doctor Nixon's request I examined
the patient, and, by his kind permission, learned the following
particulars respecting him.   He had always been delicate, and
had rheumatism three years previously; was again attacked with
pain and swelling of the small joints of the feet a few days prior
to admittance.   The disease, as commonly happens in such cases,
migrated to the hands, and the patient was suffering acutely
when I visited him with Dr. Nixon.   The pulse was rapid, but
regular; the superficial epigastric veins engorged and tortuous,
but no venous congestion of the neck; precordial dulness normal
in extent; the heart's action tumultuous, but its sounds normal,
with this exception, namely, that in the second intercostal space
of the left side, close to the sternum, and over a space equal in
size to the bell-end of the stethoscope, a sharp grating noise
accompanied the second sound; a fremitus was likewise per-
ceptible in the same situation; but neither phenomenon was
elsewhere discoverable.

Having never previously heard a single pericardial friction-sound of *diastolic* rhythm, I expressed the opinion that the sound in question was an example of the exceedingly rare phenomenon, a regurgitant murmur in the pulmonary artery.

In the course of a few days loud and characteristic pericardial friction-sound of double rhythm was audible throughout the precordium, and no doubt could exist as to the nature of the case. Effusion rapidly set in, and the patient for several days was on the point of death; but, under the judicious treatment of Dr. Nixon, with a mild preparation of iron, and an ample allowance of wine, the liquid effusion was gradually dispersed, the double *frottement* returned, and ultimately ceased, the superficial venous engorgement of the abdomen disappeared, and the patient, restored to comparatively good health, was discharged.

I did not at the time venture upon an explanation of the localized venous engorgement of the abdominal wall; nor have I since been able to suggest a plausible solution of the difficulty. The strictly diastolic rhythm of the localized pericardial friction-sound at the outset, constitutes, however, the principal feature of interest in this case; it is the only example of the kind I have met with. The error of diagnosis arising from the rarity and eccentric character of the phenomenon, which the harsh quality of the sound should have prevented, was quickly and rudely exposed in the progress of the case.

CASE XIX.—*Rheumatic Endo-pericarditis; Recovery.*

Matthew R., aged thirty-three years, carpenter, intemperate, admitted March 11th, 1872, for subacute rheumatism engaging the smaller joints of the feet and hands, and migratory in character. He was thin, pale, and weak, suffering much from articular pain, which deprived him of sleep, but was not accompanied by swelling or cardiac complication. The treatment consisted in large doses (fifteen and thirty grains respectively) of bicarbonate and acetate of potash, with five drops of tincture of aconite, repeated every second hour: it was not, however, attended with much benefit. Opium was subsequently given in gr. j doses every fourth hour, with decided relief of pain; and the affected

joints were stuped with warm laudanum, and wrapped in carded cotton

March 25th. Pulse 96, soft, and regular ; some blood in sputa ; base of left lung congested, and loud sawing *frottement* audible all over precordium and left front ; also towards the right side, as far as the right sterno-clavicular articulation, but not in the neck or posteriorly ; it is double, corresponding to the sounds of the heart, but audible during expiration only ; no *fré-missement*, or carotid pulsation ; stabbing pain at the apex of the heart ; breathing tranquil, and 24 in the minute. Precordial pain was entirely relieved by the application of two leeches, fol-lowed by a warm poultice. Calomel was given in doses of gr. j every hour. On the following day a mitral systolic and sub-stitutive murmur existed at the apex ; and on the 27th, a single friction-sound was audible at the left base, and at the apex a double *frottement*, through which the soft systolic mitral murmur was distinguishable Pulse 108. No increase of precordial dul-ness. This man was discharged in the first week of April quite free from pain No precordial friction-sound then existed, but the apex-systolic murmur was still distinctly audible.

CASE XX.—*Strain, followed by Pain in the chest, Dyspnœa, and Dysphagia ; Dulness on Percussion over the left side of the chest with absence of Respiratory sound and of Vocal Fremi-tus ; Displacement of the Heart to right side ; Pericardial Frottement ; Remarkable Rapidity of Pulse ; Aggravated Dyspnœa, and Orthopnœa ; Death by Asthenia. Copious Effusion of Serum into the left pleural cavity ; Cancerous Solidification of the left lung, with Adhesion to the Pericar-dium and chest-wall, and remarkable Cancerous Thickening of the left pleura ; Cancerous Transformation of the base of the right lung and right pleura in an earlier stage, with Adhesion of the Lung to the chest-wall ; Cancerous Pericar-ditis, with Partial Adhesion to the Heart, which was soft, but not otherwise diseased ; Cancer of the liver, mesentery, omenta, and left supra-renal capsule.*

Michael R., aged twenty-two, a labourer, was admitted into the Mater Misericordiæ Hospital on the 2nd of January, 1866. He

had enjoyed general good health up to July of the preceding year, when, on pushing from him with both his hands a large clump of turf, he experienced in the region of the heart a sensation as if something had burst, followed by a feeling of scalding and tightness, and by shortness of breath; his head "swam," his sight became dim, and he was obliged to sit down. After a few hours he recovered sufficiently to be able to go home. This feeling in the region of the heart continued some days; it then passed away, and his breathing was so much improved that he was able to resume his work. He did not, however, feel quite well.

About three months subsequently he felt some difficulty in swallowing. The morsel in its descent was stopped behind the lower end of the sternum and returned some distance, but after an effort it passed onwards into the stomach. After swallowing a few mouthfuls in this way the food passed with less difficulty. Liquids passed with facility, but he could not swallow solids without taking a mouthful of drink.

About this time, also, he caught cold whilst attending his mother in her last illness, and had cough, muscular pains in the neck and back, and stitch in the left side. Whilst journeying to town on the preceding day he became jaundiced in the train When admitted, he was of a deep lemon-colour, and the urine was loaded with bile-pigment. Dysphagia as above described. Pulse 108, and feeble, but regular; respiration 36; tongue slightly coated; orthopnœa; liver prominent, especially the left lobe, tender to pressure, and projecting two inches below the costal cartilages; bowels constipated. Left side of chest universally and absolutely dull, and on this side no respiratory sound audible except posteriorly, above the angle of the scapula, where it was faintly bronchial, and where likewise the voice was ægophonic; vocal vibration was abolished on this side, which in girth exceeded the right by half an inch, was smooth by obliteration of the intercostal spaces, and motionless during respiration. Behind the anterior fold of the axilla some distended superficial veins were visible. The heart pulsated strongly behind the right margin and lower portion of sternum; its sounds were clear and unassociated with murmur. Preceding the first sound, however, an obscure grating, regarded as pericardial, was

audible. On the following day this grating sound was some-what "to-and-fro" in character. The pulsations of the heart were heard all over the chest, but were especially loud over the left side, and were transmitted, but not with intensity, in the course of the aorta. Over the right side resonance was clear, and respiration loud.

Six leeches were applied to the epigastrium, followed by a poultice ; and two grains of mercury with chalk, and an equal quantity of James' powder, were given every third hour.

January 6th. Pulse 162, weak, and intermitting ; respira-tion 42.

7th. Cannot lie down ; dyspnœa very urgent ; takes rest by leaning over on a pillow placed upon a table to his left ; no lividity of surface ; crepitus heard in right mammary region ; respiration and resonance elsewhere normal on that side ; peri-cardial *frottement* no longer audible. To have a terebinthinate enema, and, every fourth hour, a draught composed of sulphuric ether, 3ss, and sedative liquor of opium, ℞ xv.

8th. Dulness over base of right lung posteriorly, and friction in mammary region, where crepitus had been previously heard. Up to this date the diagnosis was by no means clear. The his-tory of the case, and the existence of dysphagia were suggestive of aneurism ; but there were obvious and insuperable objections to this view.

9th. Pulse 174 ; respiration 42 ; great respiratory distress, and fulness with pain in epigastrium.

13th. Has improved under the use of mercurial inunction, aperient enemata, blisters applied over the chest and epigas-trium, and repetition of anodyne draughts. Can now swallow solids in small morsels. Heart's sounds most distinctly audible over midsternum, where a soft bellows-murmur preceded the first sound, but fell short of it. No lividity or œdema ; jaundice continued.

15th. Is sinking Voice nasal, and diptheritic deposit on inside of cheeks. Died at 8 P.M.

Body examined fourteen hours after death ; was rigid, and not much emaciated.

Abdomen. Peritoneum greatly thickened, and opaque ; liver

much enlarged, olive-coloured, and exhibiting several large can-
cerous nodules upon its surface, and throughout its substance ;
gall-bladder large and distended with bile ; pancreas had been
converted into a large nodulated scirrhus mass, involving the
common biliary duct. The mesentery and omenta were pervaded
by cancerous nodules, varying in size from that of duck-shot to
that of a musket-ball. Right supra-renal capsule attached to
liver, and in a state of cancerous transformation. Left capsule,
both kidneys, spleen, and hollow viscera, healthy.

Chest. Left pleural cavity full of straw-coloured serum, and
pleura, both parietal and pulmonary, greatly thickened and
roughened by cancerous nodules and effused lymph, the latter
quite as thick as upper-leather. Both lungs were attached to the
walls of the chest by adhesions which, posteriorly, were remark-
ably strong. The anterior edge of the left lung was incorporated
with the pericardium by thickened pleura, which here was as
thick as sole-leather. On section, the lung was found to have
been converted into a mass of scirrhus, it was solid, rigid, and
heavy. The base of the right lung was similarly altered, but in
an earlier stage ; the pulmonary pleura was thickened, and from
its deep surface thick bands passed into the substance of the
lung, branching, and dividing the surface of section into poly-
gonal areolæ, in which healthy and crepitant lung tissue was
lodged. The apex of the right lung was apparently sound, and
was resonant and crepitant. The surface of the heart was rough
and granulated, it was of a dark red colour, and attached to the
pericardium by reticulated bands of adhesion, which were easily
broken down, and obviously recent. The heart was soft, and
the valves were all in a healthy state.

CHAPTER VI.

DISEASES OF THE SUBSTANCE OF THE HEART.

DISEASES of the substance of the heart may be conveniently divided into:

1. Morbid changes of volume.
   - (a) Hypertrophy.
   - (b) Atrophy.
   - (c) Dilatation.
   - (d) Aneurism.
2. Changes in consistence.
   - (a) Softening (inflammatory, typhoid, fatty).
   - (b) Induration.
3. Adventitious products.
   - (a) Adipose (fatty accumulation, fatty substitution, fatty transformation).
   - (b) Hydatids.
   - (c) Fibroid growth (hyperplasia).
   - (d) Osteoid growth.
   - (e) Tubercle.
   - (f) Cancer (medullary, scirrhus, melanotic).
   - (g) Syphiloma.
4. Wounds of the heart.
5. Phlegmasiæ.
   - (a) Myocarditis.

Hypertrophy of the heart is due essentially to an effort of compensation. The heart being in a state of healthy nutrition, it suffers, owing to one or more of the causes to be presently enumerated, from a derangement of the normal equilibrium between its dynamic capacity, as the central organ of the circulation, and the conducting properties of the vascular system.

Bertin describes hypertrophy and atrophy as the result of an "augmentation" and a "diminution" of nutrition, respectively;

whilst induration and softening he regards as "alterations" of nutrition.[*]

Lebert defines hypertrophy, in general, as "an augmentation of the essential constituent molecules of a tissue or organ, a veritable increase of nutrition, with excess of its materials."[†] As regards the heart, Rindfleisch understands hypertrophy to be "an increase of the volume of the heart, which has its foundation in a hyperplasia of the myocardium."[‡]

According to the last named writer, therefore, hypertrophy of the heart has always an inflammatory, and therefore a morbid origin This doctrine may, however, be questioned on two grounds; namely, the presence, by universal admission, of the products of inflammation in hypertrophied hearts only exceptionally; and the strict proportion observed, as in all other examples of muscular hypertrophy, between the increase of development of the heart and the augmentation of its function, in every case, without exception, of hypertrophy of that organ.

Hypertrophy of the heart may be either general or partial; and, being the result of an effort of compensation, it implies either that the portion affected, if in a healthy state, has undergone simple increase in the thickness of its walls, proportionate to the difficulty imposed upon it in carrying on the circulation; or that, being incapacitated for such increase beyond a certain limit, by malnutrition, or degenerative change, it has yielded to the obstacle opposed to it, and become also dilated.

Hence there can be only two forms of hypertrophy; namely, the *simple* form, in which the walls are thickened without alteration in the size of the cavity; and the *excentric* form, in which the walls are thickened and the cavity dilated.

To these two varieties of hypertrophy, Bertin§ has added a third, which he names *concentric*. In this, the walls are thickened, and the cavity is reduced in capacity. Hope recognizes, |

* *Maladies du Cœur*, p 345.
† *Traité d'Anatomie Pathologique*, 1857. tom. i., p. 85.
‡ *Text Book of Pathological Anatomy*. 1872, p. 226.
§ *Mem de l'Académie Royale des Sciences*, 1811 ; *Traité des Maladies du Cœur*, 1824, p. 233.
‖ *The Diseases of the Heart and the Great Vessels*, third edition, 1839, p. 234.

as the equivalents of the foregoing, three forms of hypertrophy, namely :

1. Simple hypertrophy.
2. Hypertrophy with dilatation (active aneurism of Corvisart, excentric or aneurismal hypertrophy of Bertin).
3. Hypertrophy with contraction (concentric hypertrophy of Bertin).

Of the second or *excentric* variety, two forms have been recognized; namely (*a*) that in which the walls are thickened and the cavity dilated; and (*b*) that in which the cavity is dilated, but the walls retain their natural thickness.

This latter form Hope recognizes under the designation of "hypertrophy by increased extent of the walls;" and Latham by the more simple and appropriate title of "proportionate"[*] hypertrophy. But inasmuch as the state of hypertrophy is constituted essentially by thickening of the walls, I cannot admit this sub-variety, in which that condition is absent. I prefer including it in the category of dilatations, under the title of the *simple* form of that condition of the heart. The states of hypertrophy and dilatation being relative, must be judged by reference to a fixed standard of comparison.

Laennec roughly estimated the volume of the heart, the thickness of its walls, and the capacity of its chambers, absolutely and relatively, as follows:—"The heart, comprising the auricles, ought to have a size equal to, a little less, or a very little larger than, the fist of the subject. The walls of the left ventricle ought to have a thickness a little more than double that of the walls of the right; they ought not to collapse when an incision is made into the cavity. The right ventricle, a little larger than the left, and having larger columnæ carneæ notwithstanding the inferior thickness of its walls, ought to collapse when an incision has been made into it."[†]

Bouillaud's measurements, though admittedly based on insufficient data, may be regarded as approximately correct.[‡]

He estimated the volume, weight, and thickness of the heart

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[*] *Lectures, etc., comprising Diseases of the Heart*, 1846, vol. ii., p. 182.
[†] *Opus citat.*, vol. ii., p. 404.
[‡] *Vide antea*, p. 40, for Table of Comparative Measurements, etc., relating to normal heart.

in the young and in the adult subject respectively under three heads,* viz. :

1. Normal heart.
2. Atrophied heart.
3. Hypertrophied heart.

Of the normal heart

				Ozs.	Drms.
The maximum weight is	...	...	...	11	0
The minimum ,,	...	...	...	6	2
The mean ,,	...	...	...	8	3

The average weight of the heart in males, between the ages of twenty-five and sixty years, he estimates at eight to nine ounces; and between sixteen and twenty-five years, one to two ounces less. In persons very large and strong, it might amount to eleven ounces without transgressing the limits of health. In females, the weight of the heart is less than the above by an undetermined quantity.

Circumference of the normal heart at the base of the ventricles :

				Inches.	Lines.
Maximum	...	...	...	...	10	6
Minimum	...	...	...	...	8	0
Mean ...	...	...	...	...	8	9

Length from root of aorta to apex :

Maximum	...	...	...	...	4	0
Minimum	...	...	...	...	3	$2\frac{1}{2}$
Mean ...	...	...	...	...	3	$7\frac{1}{3}$

Transverse diameter a little below base of ventricles :

Maximum	...	...	...	...	4	6
Minimum	...	...	...	...	3	5
Mean ...	...	...	...	...	3	$7\frac{1}{2}$

Thickness of walls of left ventricle at base :

Maximum	...	...	...	...	0	8
Minimum	...	...	...	...	0	5
Mean	...	...	...	...	0	6

Right ventricle at base :

Maximum	...	...	...	...	0	$3\frac{1}{2}$
Minimum	...	...	...	...	0	$1\frac{1}{2}$ to 2
Mean ...	...	..	...	...	0	$2\frac{2}{3}$

* *Traité Clinique des Maladies du Cœur*, 1835, tom. i., Prolégomènes, p. 3-4.

A general estimate of the thickness of the ventricular walls would yield,

	Lines.
For the left ventricle	7
For the right ventricle	2½

From sixteen to forty years, the thickness increases, being greater in persons of strong build and tall, than in those of opposite physique.

Thickness of interventricular septum is 11 lines.

Thickness of walls of left auricle:

	Lines.
Maximum	2
Minimum	...¾ to 1
Mean	1½

Thickness of right auricle:

	Lines.
Maximum	1¼
Minimum	¾
Mean	1

As to capacity, each of the ventricles would contain a pullet's egg, but the right somewhat exceeds the left in size. The auricles are equal in capacity respectively to the corresponding ventricles, the right somewhat exceeding the left.

Clendinning, as quoted by Hope,* gives the following as the average weight of the heart in the male and female respectively, from fifteen years upwards:

	Males.	Females.
15 to 30 years	8¼ ozs.	8½ ozs.
30 to 50 „	8½ „	8¼ .,
50 to 70 „	9½ ,,	8 „
70 and upwards	9¾ „	8 „

	Atrophy.	Normal State.
Maximum weight ...	200 grammes	350 grammes.
Minimum „ ...	135 „	200 ,,
Mean „ ...	175 ,,	262 „

* *Opus citat.*, p. 237.

		Hypertrophy.	Normal state.
Maximum weight of heart	.	688 grammes	850 grammes.
Minimum ,,	..	338 ,,	200 ,,
Mean ,,		473½ ..	262 ,,

Thus, the heaviest heart which Bouillaud has met with weighed only twenty-one and a-half ounces.

Lobstein has seen one of thirty-two ounces.

The heaviest heart which I have met with in my own practice weighed only thirty-two and a-half ounces; but I have seen and preserved one obtained from a patient of my colleague, Dr. John Hughes, which weighed forty-two ounces.

Hope mentions one which he had seen of forty-four ounces in weight.*

Doctor Stokes, as already mentioned, exhibited at the Pathological Society a heart which had been the subject of valvular disease and adhesion of the pericardium, yielding the enormous weight of four pounds and two ounces, avoirdupoise Allan Burns adduces the authority of Lieutaud for the description of a heart which weighed five pounds; and he mentions an example of simple hypertrophy of "several pounds weight," which he had himself seen.†

Thus, it is manifest that the maximum weight of an hypertrophied heart witnessed by Bouillaud has been greatly exceeded Nor can I admit his assertion that a heart of eleven ounces, although close on the confines, is not within the limits of morbid growth. I am willing to believe that in a strong man of very active habits, a heart of eleven ounces may consist with a state of apparent health, the enjoyment of society, and even a continuance, without abatement, of active duties and indulgences.   But, in such a case, an attack of serious illness of any kind, especially one of an acute febrile character, will show, by the tumultuous action of the heart, and by its early failure, that the condition of that organ, antecedent to present illness, was not that of health.

* A Treatise on the Diseases of the Heart and Great Vessels, 1839, p. 237.
† Observations on some of the Most Frequent and Important Diseases of the Heart, etc, 1809, p 40.

Hypertrophy of the chambers of the heart may be *primary* or *consecutive*, and due to :

1. Mechanical obstruction to the circulation, arising from
   (*a*) Obstruction at valvular orifice in front.
   (*b*) Incompetence of valves in front.
   (*c*) Incompetence of valves behind (mitral).
   (*d*) Rigidity of main arteries.
   (*e*) Spasmodic resistance of small arteries.
2. Habitual over-exertion of the voluntary muscular system.
3. Habitual nervous excitement of the heart.

The two last mentioned causes (Nos. 2 and 3) are alone capable of producing primary or uncomplicated hypertrophy of the heart; but, inasmuch as hypertrophy without complication, causative of, or dependent upon it, is never fatal, the opportunity for examining a heart affected with this form of hypertrophy, unassociated with antecedent or consecutive disease, is presented only where death in such a case is caused by acute febrile disease, or by accident.

The changes consecutive to hypertrophy of the heart, and in the order of their frequency, are,

1. Tissue degeneration of the heart, granular and fatty.
2. Dilatation of either ventricle, or of both ventricles.
3. Atheroma of the aorta and its branches.
4. Dilatation of the aorta, and consequent inadequacy of the aortic valves.
5. Fatty degeneration of the kidneys.
6. Calcareous transformation of the aortic valves.

Hypertrophy of the heart from severe and long-continued mechanical labour is the result of a compensatory effort on the part of that organ ; additional duty has been imposed upon it ; its rate and force of contraction have been proportionately increased, and so long as its nutrition may be quickened in an equal ratio, so long will increase in the thickness of its walls keep pace with, and strictly counterbalance exaggerated function. Ultimately, however, owing to impairment of primary digestion, or of assimilation, nutrition fails, or rather, it is no longer capable of being maintained at this high standard ; degenerative changes in the tissue of the heart are now set up ; its

power of resistance to excentric pressure is proportionately les-
sened, and dilatation follows.   Concurrently with degeneration
of the heart, but oftener consecutively to it, atheromatous change
in the coats of the aorta and smaller arteries takes place, and
the kidneys undergo the fatty change of structure.   The period
occupied by these successive processes may be very considerable,
varying from six months to as many years ; and should the case
in its complex form be now for the first time presented to a
physician, it would be difficult indeed to determine with cer-
tainty the order of sequence of the several changes indicated.

I have no doubt whatever that hypertrophy of the heart, ari-
sing primarily from excessive muscular exertion, or from habi-
tual emotional excitement, whether of joy, fear, anxiety, or anger,
but ultimately associated with renal disease by general progres-
sive degeneration of tissue, has been repeatedly set down as a
consequence of the renal affection.

Doctor Quain* classifies all cases of hypertrophy of the heart
under three heads, according to the tissue primarily and prin-
cipally involved ; namely, hypertrophy of,

1. The muscular tissue.
2. The connective tissue.
3. The adipose tissue.

The *causes* of hypertrophy he regards as of three kinds, viz.,

1. Nervous.
2. Mechanical.
3. Nutritive.

The first is exemplified in the primary enlargement so com-
mon in periods of popular excitement.   The second class of
causes may be located in the heart itself, in the great vessels,
or in the small and distant vessels, as exemplified in Bright's
disease, and in pregnancy.

The causes which operate through nutrition may be general
(anæmic) or local (inflammatory) ; they are exemplified respec-
tively in the enlargement of anæmia, which, however, is usually
the result of dilatation rather than hypertrophy ; and in that
consecutive to pericarditis, and to inflammation of the con-
nective tissue of the heart.   In both these latter instances the

* Lumleian Lectures, *Medical Times and Gazette*, March 23rd, 1872.

enlargement is due to hypertrophy of the interstitial fibrous tissue of the heart, and constitutes a veritable hyperplasia; it is remarkably prone to changes of degeneration.

In connective tissue hypertrophy, Quain remarks, the walls are dense and leathery, and the colour of various tints, from that of muscular fibre to a light grey hue.

Under the microscope fibrous tissue is seen in all stages of development between, and pressing upon, the muscular fibre

Corvisart long since described this form of hypertrophy of the heart, and attributed it to inflammation of the connective tissue of the organ.[*]

Rokitansky even doubts the increase of muscular substance in any form of hypertrophy, and intimates that, in his opinion, the increased bulk is due in all cases to excessive development of the areolar tissue of the organ.[†]

Laennec, Sir W. Jenner, Williams, and Ormerod, have likewise recognized hypertrophy of the areolar tissue of the heart

Nevertheless, I doubt that it constitutes a form of hypertrophy involving increased volume of the heart. Hypertrophy of the connective tissue of other organs, the liver, the spleen, and the lungs, leads to atrophy and general diminution of volume, not to hypertrophy of their parenchyma. I have not met with an example of hypertrophy of the heart, excepting those in which its bulk was increased from superficial deposit of fibrin, the result of repeated accessions of pericarditis, in which increased development of connective tissue was the cause of augmentation of its volume. It is theoretically conceivable that plastic exudation, following close upon inflammatory irritation of the fibrous tissue of the heart, might give rise to enlargement of the whole organ. But even in passive or glandular organs, as is well known, such effect, though actually produced, is only temporary, atrophy and induration being consecutive and proximate changes. In the heart, subject as that organ is to the operation of high pressure from within, repeated fifty to eighty times or oftener every minute, and tending to stretch its walls and di-

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[*] *A Treatise on the Diseases and Organic Lesions of the Heart and Great Vessels* translated by Hebb, p. 56.

[†] *Pathological Anatomy*, Sydenham Society's edition, vol. i., p. 40.

late its cavities, hyperplastic increase of the fibrous element, by lessening the resisting power of its walls to internal pressure, determines, even more rapidly than in other organs, atrophy of substance, and dilatation of the parenchymatous recesses or cavities of the organ.

Rindfleisch even maintains that inflammation of the connective tissue of the heart, by extension from the endocardium, and limited to a small space, is the primary cause of partial aneurism of the heart, by determining thinning of its walls.

Fatty hypertrophy differs from fatty degeneration, as consisting in accumulation of fat beneath the pericardium and between the muscular fibres.

As *causes* of hypertrophy, Corvisart mentions all such as give rise to obstructed circulation ; moral causes ; severe labour or exercise ; and, " perhaps," the stimulant quality of the blood.

To the preceding causes Bouillaud adds chronic inflammation of the pericardium or endocardium.

Arising from the former causes, hypertrophy would be "primitive," according to his nomenclature ; and arising from the latter it would be " consecutive."

Hope, Lebert, Quain, and indeed most modern authorities, likewise hold that inflammation is a direct and common cause of hypertrophy.

" Concentric hypertrophy," as first noticed and described by Bertin, has been likewise admitted by Bouillaud, who gives three cases in illustration,* by Laennec,† Hope,‡ Rokitansky,§ and Lebert.‖

For the reasons previously stated, I venture to question its occurrence as a distinct pathological change ; and further, because I have not met with a single example of the condition of the heart corresponding to the "concentric hypertrophy" of Bertin, that would not admit of the explanation of Cruveillier previously referred to, namely, thickening of the walls and reduction of the cavities of a heart in a state of simple hypertrophy, by spasmodic contraction at the moment of sudden

* *Opus citat.*, tom ii., observations 118, 119, 120.
† *Traité d'Auscultation Médiate*, 1826, tom. ii., p. 412.
‡ *Opus citat.*, p. 234.
§ *Pathological Anatomy*, vol i., p. 156.
‖ *Traité d'Anatomie Pathologique*, 1857, tom i., p. 85.

death; or which was not an example of atrophy with contraction of the cavities.* In this opinion I am fortified by the observations of Dr. George Budd, who states that of eight cases collected by him, exhibiting the condition of heart regarded as peculiar to concentric hypertrophy, not one presented signs during life, or *post mortem* evidence of obstructed circulation through the heart. There was no irregularity of pulse, no dropsical effusion, and after death the right chambers of the heart were found not dilated. He adds, with much force, that it is impossible to conceive that a left ventricle, not capable of containing an almond, should present no obstacle to the circulation during life.†

In Laennec's case the walls of the left ventricle were one and a-half inch thick, and its cavity not large enough to contain an almond stript of its shell; yet, the day preceding death the breathing was free, and "nothing (he says) led me to suppose that this man had a disease of the heart."

Bertin gives the particulars of four cases of concentric hypertrophy.‡ In the first the pulse was regular, small, hard, and strong; the pulsations of the heart strong and concentrated, and the sounds dull and seemingly remote. On examination of the body the walls of the left ventricle were found an inch thick in the middle, the fleshy columns thickened, the cavity much diminished, the aorta reduced in calibre, and scaly, and the other chambers and valves sound. There was softening of the brain, and extensive disease of the arteries generally.

In his second case, there had been great embarrassment of breathing. The right chambers were thin and dilated, the left auricle normal, the wall of left ventricle one and a-half inch thick near the base, and its cavity reduced to half its normal capacity; the aorta was atheromatous.

In his third case there was likewise great oppression of breathing. On examination after death the left ventricle was found double its natural thickness, and its cavity reduced to the size of an ordinary filbert. The other cavities, and all the valves, were in a healthy condition.

* *Dictionnaire de Médecine et Chirurgie Pratique.* Article "Hypertrophie."
† *Medico-Chirurgical Transactions*, vol. xxi., 1838.
‡ *Opus citat.*, p. 310, observations 80, 81, 82, 87.

The fourth case was an example of concentric hypertrophy affecting the right ventricle only. Its walls were eleven to sixteen lines thick, and its cavity reduced to the size of a pigeon's egg. The foramen ovale was patent, and the pulmonary artery nearly closed by a diaphragm.

In the first of the cases just given in summary, the diseased state of the aorta and of the arteries generally, was sufficient to account for hypertrophy of the left ventricle, which was probably in the simple form during life; a view seemingly confirmed by the character of the pulse, and of the heart's action.

In the second case, the atheromatous condition of the aorta would imply a state of hypertrophy, simple or dilated, of the left ventricle, and the dyspnœa would be, to my view, satisfactorily accounted for by the atheromatous state of the aorta, and the attenuation of the right ventricle. In the third case details are wanting, e.g., as to the period during which dyspnœa had existed. Otherwise this would seem the only one of Bertin's cases which might be adduced with effect, in support of his doctrine of the existence of concentric hypertrophy as a primary pathological alteration; as the other cavities and all the valves of the heart were sound.

Bertin explains hypertrophy of the right ventricle in his fourth case by reference to the entrance of arterial blood through the foramen ovale, from the left to the right auricle, and consequent " nutritive irritation " of the walls of the right ventricle.

But it may be remarked that such cause should give rise likewise, and in the first instance, to hypertrophy of the right auricle, which, however, did not exist. Theoretically, it would seem that for the production of a state of concentric hypertrophy of the right ventricle three conditions should coexist; namely, a direct passage of blood from right to left auricle, through an open foramen ovale; competency of the tricuspid valve; and contracted pulmonary orifice, by which an obstacle is opposed to the escape of blood from the right ventricle.* The authority of Bertin is entitled to so much respect in con-

* An example of contracted pulmonary artery is given by Morgagni, *De Sedibus et Causis Morborum*, tom. ii., epistol. xvii., p. 525.

nexion with any subject of cardiac pathology, that I have felt it necessary to explain at length my reasons for dissenting from him on this subject

There is much disagreement amongst pathologists as to the most common form of hypertrophy. Corvisart considers that with dilatation or "active aneurism," the usual form.

Rokitansky declares that simple hypertrophy is rarely met with, and oftener in the left than in the right ventricle; it is, according to him, a transitional state, and merges into the excentric form, which is, *par excellence*, the typical form of hypertrophy. The side affected is most frequently the left, whence the pathological change extends to the right, and when both are affected, the condition constituting *cor tauranum* is established. When the auricles are affected with hypertrophy, the change is due to contraction of the corresponding auriculo-ventricular orifice.*

I am of opinion that hypertrophy is, in the great majority of instances, associated with some degree of dilatation

In this opinion I am sustained by the recent authority of Rindfleisch, who goes the length of declaring that "a certain degree of dilatation is associated with every hypertrophy of the heart"† Whilst maintaining that hypertrophy of the heart is most frequently presented under the excentric form, I cannot subscribe the exclusive doctrine of Rindfleisch. Simple hypertrophy is certainly, and not unfrequently, met with; but always, as Rokitansky asserts, as a state of transition from health to dilated hypertrophy. It is, *par excellence*, the condition in which the left ventricle is found in the early stage of hypertrophy from renal disease; the stage, namely, of considerable duration, which precedes nutritive decay and tissue degeneration of the organ When these changes have set in, as they invariably do in the ordinary course of pathological sequence, the ventricle undergoes dilatation, usually in excess of antecedent hypertrophy, and excentric hypertrophy of a degenerated left ventricle is the result. But, inasmuch as the impairment of general nutri-

* *Opus citat.*, vol. n., p. 156-7.
† *Text-Book of Pathological Anatomy*, translated by Kloman and Miles, 1872, p. 227.

tion causative of, or attendant upon renal disease, must operate upon the entire heart, the right ventricle likewise suffers ; but not having previously undergone hypertrophy, the condition in which it is found is usually that of dilatation with thinning of the walls, and granular or fatty degeneration of the muscular fibre.

Walshe states that general hypertrophy of the heart is always of the excentric kind, and that the individual chambers are affected in the following order of frequency : viz., left ventricle, left auricle, right ventricle, and *longo intervallo*, right auricle.[*]

Hypertrophy, according to Stokes, is rarely confined to one cavity[†]   But hypertrophy of the left ventricle of renal origin, where the valves are free from disease, constitutes an exception to this statement.   The other chambers undergo no change till tissue-decay and dilatation of the left ventricle have set in, as they do in ordinary pathological succession ; and then, partici- pating in the effects of malnutrition, and influenced by the pro- gressive incapacity of the left ventricle in front, they undergo gradual dilatation of the atrophic or attenuative kind.

Mechanical obstruction to the circulation, of whatever kind, was the only cause of hypertrophy ("active aneurism") recog- nized by Corvisart.   For the right auricle, the obstacle must be located at the tricuspid orifice; and for the right ventricle, at the orifice of the pulmonary artery, or in the lungs.   For the left auricle the cause of obstruction must be located at the mitral orifice ; and for the left ventricle it may consist in valvular obstruction at the mouth of the aorta, tortuosity of the arteries, dropsical effusion, or capillary obstruction by spasm of the skin.[‡]

The last mentioned cause is of special interest to modern pathologists, as indicating on the part of Corvisart, a knowledge of the connexion, at least in their remote relationship, between renal disease and hypertrophy of the left ventricle of the heart. A rigid, pallid, unperspiring state of the skin, constitutes one of the earliest and most suggestive of the general symptoms of

[*] *Opus antea citat.*, p. 277-8.

[†] *The Diseases of the Heart and Aorta*, p. 273.

[‡] *On the Diseases and Organic Lesions of the Heart and Great Vessels*, translated by Hebb, p. 107.

renal disease. The cause of this state of the integument was unknown to Corvisart; yet the condition itself, and its frequent association with tumultuous action and enlargement of the heart, did not escape his observation. "Active aneurism," according to him, is more common in the left ventricle than in the right, because the former is more irritable, and under a stimulus, more susceptible of active nutrition; and finally, because obstacles to the circulation are of more frequent occurrence at the orifice of the aorta than at that of the pulmonary artery.* Causes which give rise to "active aneurism" of the left ventricle, usually produce, consecutively, "passive aneurism" of the right.

Singularly enough, whilst asserting that mechanical obstruction is the only cause of hypertrophy, he propounds the paradox, that in that condition of the heart the increased power is in excess of the resistance to be overcome.

Bouillaud,† in addition to the foregoing, recognizes moral agency in the production of hypertrophy. He likewise mentions as causes, severe and long continued bodily labour or exercise, and chronic inflammation of the pericardium and endocardium. As already stated, he designates hypertrophy arising from the last mentioned cause as "consecutive," and that produced by all those previously mentioned, as "primitive." He gives thirty-three cases of chronic peri- or endocarditis, in all of which hypertrophy existed; but he does not inform us whether valvular disease was likewise present in these cases; an omission which renders his premises of little value in regard to the conclusion he desires to draw from them. Amongst the possible causes of hypertrophy of the right ventricle, he includes the entrance of blood from the left ventricle, through a patent foramen ovale, or perforated ventricular septum, on the principle of "excitation," and gives the particulars of four cases,‡ in each of which the twofold lesion existed. The pathogenic connexion, however, he considers as "somewhat hypothetical."

Lebert mentions, amongst the remote causes of hypertrophy of the heart, the tubercular diathesis, and gives a case in illus-

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* *Opus citat*, p. 66.
† *Traité Clinique des Maladies du Cœur*, 1835, tom. ii., p. 365, *et suivant*.
‡ Observations 75, 76, 77, and 79.

tration, in which, however, tubercular pericarditis had been set up.[*]

Of one hundred and seventy-one cases of phthisis collected by Quain, the heart was enlarged in 25 66 per cent. in males, and only in 7 0 in females. In males the heart was small in 53 per cent., and in females in 67 per cent. In males it was normal in 21 per cent., and in females in 26 per cent. In a girl of eight years, the heart weighed only two ounces; and in another, aged fourteen years, it weighed only one ounce and fourteen drachms.[†] It is not stated in what proportion, if any, of the cases in which hypertrophy existed, other causes of enlargement were present.

I have not seen hypertrophy of the heart in bodies exhibiting evidence of extensive tubercular development, or advanced pulmonary phthisis, in which one or more of the ordinary causes of hypertrophy did not likewise exist. These causes, too, renal or cardiac, judging from the stage to which they had attained, as well as from the history of the patients' last illness, were anterior in date to the phthisical affection

Hermann Lebert has recently expressed the opinion, in support of which he gives several illustrative cases, that in relation to pulmonary tuberculosis, diseases of the heart and aorta, such as are capable of giving rise to impairment of general or special nutrition, stand in the position of cause, not effect [‡]

Rokitansky goes the length of declaring hypertrophy and dilatation of the heart to be incompatible with tuberculosis; atrophy being the usual condition of the heart met with in that state [§] He justly attributes hypertrophy with dilatation of the right chambers in many instances to curvature of the spine and pulmonary emphysema, [||] and mentions, as producing a similar effect, narrowing of the mitral orifice.

Obstruction is, *par excellence*, the cause of hypertrophy in the

[*] *Traité d'Anatomie Pathologique*, 1857, tom. 1 , observation clxiv , p, 555

[†] *Loco citat.*

[‡] *Medical Times and Gazette*, December 11th, 1869

[§] *Pathological Anatomy*, vol 1 . p. 165 and 316

[||] *Opus citat* , vol. i p. 163 4.

opinion of Hope, whilst constant congestion of the cavities is, in an equal degree, the cause of dilatation.*

Walshe classifies the causes of hypertrophy under seven heads, viz ,†

1. Causes originating in the system.
    (*a*) Excessive nourishment, especially nitrogenized, with stimulants and sedentary habits.
    (*b*) Excessive exercise, *e.g.*, walking, rowing.
2. Causes originating in the blood.
    (*a*) Uræmia (?).
    (*b*) Rheumatic hyperinosis (?).
3. Causes originating in the heart itself.
    (*a*) Functional excitement, as in prolonged anæmia, and chronic pericarditis.
    (*b*) Mechanical obstruction; (affecting left ventricle , aortic constriction, aortic regurgitation, mitral regurgitation, mitral constriction (?), mitral regurgitation and constriction combined; (affecting right ventricle), tricuspid regurgitation, pulmonary constriction, mitral regurgitation through engorgement of lungs; (affecting left auricle), mitral regurgitation and constriction, aortic regurgitation and constriction, but in much less degree; (affecting right auricle), tricuspid regurgitation.
4. Causes originating in the great vessels.
    (*a*) Pressure obstructing their interior.
    (*b*) Smallness or constriction of aorta.
    (*c*) Aneurism of aortic arch near the heart (?).
    (*d*) Diminished elasticity of the coats of the aorta or pulmonary artery, affecting left or right ventricle.
5. Causes originating in the lung-circulation, and affecting right ventricle.
    (*a*) Chronic bronchitis.
    (*b*) Emphysema.
    (*c*) Contraction after pleurisy.
    (*d*) Dilatation of bronchi and cirrhosis of lung.

* *A Treatise on the Diseases of the Heart and Great Vessels,* 1839, p. 246.

† *The Diseases of the Heart,* 1863, p. 280. The notes of interrogation are in the original

(c) Diminution of cavity of chest by deformity or by abdominal tumors (?).

6. Causes originating in the kidneys.

(a) Persistent obstruction in their capillary circulation (?).

7. Causes originating in systemic capillaries.

(a) Prolonged obstruction of any given mechanism in some large portion of the systemic capillaries (?).

He doubts Bright's theory of hypertrophy in renal disease, regarding it as like the renal affection itself, "an additional local expression of the main diathesis," and as in part arising likewise from specific irritation of the blood on the endocardium.[*]

Doctor Stokes, whilst admitting hypertrophy from other causes, declares that in the absence of disease in the valves or aorta, it is exceptional.[†] But in the various forms of Bright's disease, now so commonly met with in our hospitals, hypertrophy of the heart, confined to the left ventricle, is rather the rule than the exception.

I cannot admit a special form of hypertrophy under the designation of "fatty," as mentioned by Quain, who describes the fat as mixed up with the muscular fibres throughout, but in less proportion towards the interior, and immediately beneath the endocardium limited to a few fat cells at intervals between the fibres.[‡] Fat thus deposited among the muscular fibres of an hypertrophied heart, should be regarded as the result of consecutive and degenerative, not primary or constructive changes. The only example of fatty hypertrophy which can be admitted is of a *quasi* character, namely, that of a heart magnified by deposition of fat upon the outer surface, but in reality atrophied *quoad* its muscular substance (*vide* "Aneurism," case of Mr B.).

Hypertrophy of the heart has been attributed to pregnancy by some writers. Thus, in 1828, Lacher announced, as the result of his examination of one hundred and thirty healthy women who had died in child-birth, that the heart was hypertrophied in all; the left ventricle, which was the part affected, being increased in thickness from a quarter to half an inch.[§]

* *Opus citat* , p. 284.

† *The Diseases of the Heart and the Aorta*, 1854, p. 345

‡ Lumleian Lectures, *Lancet*, March 23rd, 1872.

§ Doctor Quain's Lectures, *loco citat*

This form of hypertrophy, which has been attributed to the pressure of the gravid uterus upon the abdominal arteries, and to the increased circulation in the uterus itself, must, like the growth of the latter organ at that period, be of only temporary duration, and subject, like it, to consecutive involution.   Hypertrophy of the left ventricle in connexion with albuminuria was first explained in 1817 by the late Mr. James of Exeter, as caused by obstruction in the systemic capillaries,* and subsequently by Bright† and George Johnson.‡

Wilks, in 1853, thought it might be due to atheromatous change in the vessels, and Traube has conjectured that in renal disease obstruction in the kidneys themselves may be the cause of hypertrophy of the heart.

Doctor Johnson has announced, as the result of his recent investigations, the existence, in the granular form of renal disease, of hypertrophy of the muscular coat of the minute arteries throughout the body.   He writes : " The glandular tissues of the kidney suffer in the discharge of their excretory functions, and the organ wastes and contracts. The blood is then rendered secondarily impure by retained urinary excreta, and it is otherwise altered in its composition   This morbidly changed blood excites unusual contraction of the minute arteries, by which the circulation is impeded ; the left ventricle, on the other hand, beats with increased force to drive the blood through the resisting arteries.   The result of this antagonism of forces is, that the muscular walls of the minute arteries throughout the body, and those of the left ventricle of the heart, become simultaneously and equally hypertrophied §

This doctrine has been challenged by Sir W. Gull and Dr Sutton, in a recent communication to the Medico-Chirurgical Society of London,‖ and mainly on the evidence afforded by

* As stated by Quain, *loco antea citat.*

† *Reports of Medical Cases*, 1827, vol. ii , part i., and *Guy's Hospital Reports*, vol i., 1836

‡ *Diseases of the Kidneys*, 1852, and *Medico-Chirurgical Transactions*, passim, during the preceding six years.

§ *Medico-Chirurgical Transactions*, vol. xxxiii., and *British Medical Journal*, March 7th, 1868, and June 8th, 1872.

‖ *The Pathology and Symptoms of the Contracted Kidney, commonly called Gouty or Cirrhosed Kidney*, June, 1872.

microscopical examination of the arterial coats.   They hold that the change in the minute arteries consists in thickening of the *intima* and *adventitia*, from a deposit of " hyaline-fibroid " material in the arterioles, and " hyaline-granular " in the capillaries; which, in the intertubular and intervascular connective tissue, assumes a fibroid character.   This change induces a condition which they propose to designate " arterio-capillary fibrosis."   It takes place in the minute arteries throughout the body, but especially in those of the pia mater, and it may be unaccompanied by any change in the kidneys.   Hence, according to them, hypertrophy of the heart ; which, being due to increased muscular action of the organ, consists in increased development of its muscular structure exclusively, and differs from hyperplasia or connective tissue hypertrophy, the product of irritation, as from the excessive use of alcohol.

Thus, when the heart is found hypertrophied in connexion with chronic renal disease, the primary cause of enlargement of that organ should be sought, not in the kidneys, but in the minute arteries and capillaries, the " fibrosis " of which would constitute the *point de départ* of the series of organic changes, including that of the heart.   I am not aware, however, that any examples of cardiac hypertrophy have been adduced, which were traceable to such change in the arteries, to the exclusion of all other possible causes.*

These pathologists, in short, hold that fibroid change in the kidneys alone is not competent to produce hypertrophy of the heart ; that, where this structural change of the kidneys is primary, as in young persons, it may be fatal by uræmia, without having produced either thickening of the arterioles, or hypertrophy of the heart.†   The conclusions at which they have arrived are summarized by Sir W. Gull, as follows :‡ " That there is a diseased state coming on about the middle period of life, which is characterized by a morbid formation of fibroid, or

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* *Vide* Dr. Murchison's case, *Transactions of Pathological Society of London,* vol. xxii.

† See an example of this kind, exhibited by Dr. Murchison, and already referred to.

‡ *British Medical Journal,* December 28th, 1872.

hyaline-fibroid tissue, in the arterioles and capillaries : that this is accompanied with atrophy of the adjacent tissues in vary-ing degrees : that this morbid change commonly begins in the kidneys, but may begin primarily in other organs : that the contraction and atrophy of the kidneys is not a cause of the disease, but only part and parcel of the general morbid state : that hypertrophy of the heart is due to changes in the arterioles and capillaries, whereby their elasticity is diminished, and so the blood retarded : it is not due to a morbid condition of the blood itself : that the blood may be fatally affected by disease of the kidneys, without producing any change in the heart, provided the morbid condition of the vessels alluded to is absent : that the causes which lead to these vascular changes are not yet fully elucidated : that they have an alliance with senile conditions, though, probably, they are in their nature distinct."

This much, at least, is certain, that in the class of persons, and at the period of life when cirrhosis of the kidneys is most pre-valent, namely, in the gouty, and after the age of forty years, disease of the arteries of the first, second, and third order, con-sisting in tortuosity of the vessel and rigidity of its coats, is likewise most common ; and that with these two conditions, hypertrophy of the left ventricle of the heart, with or without valvular disease, is invariably associated.

It is no less certain that in some cases, no doubt few in num-ber, granular degeneration of the kidneys, and hypertrophy of the left ventricle, may coexist without arterial or valvular disease. This is likewise the opinion of Dr. Grainger Stewart;[*] and, further, these changes may occur at a period of life, and under such other circumstances as preclude the supposition that general arterio-capillary fibrosis was present. Case 27 (Marianne Carroll) is a good example of this kind. This girl was only nineteen years of age, and her illness dated back not more than twelve months. The kidneys were granular, weighing two and a-half, and three ounces respectively. The heart weighed nine-teen ounces, the left ventricle was greatly hypertrophied ; and there was no arterial or valvular disease save recent congestion of the mitral.

* *British Medical Journal*, November 15th, 1873.

Doctor George Johnson has, after a careful personal inspection, criticized the microscopical preparations offered in support of this theory, declaring that the alleged changes were due, not to a morbid process, but to soaking in glycerine and acetic acid. I do not think his objections have been satisfactorily answered, or that his doctrine has been in any measure disturbed by these investigations.

The condition of muscular hypertrophy was first made out in the minute arteries of the brain, which, owing to the facility presented by them for examination, from the laxity of their areolar connexions, were specially selected for this purpose; but it exists in no less degree in the corresponding vessels of other parts of the body.

Thickening of the muscular element of the walls of the systemic arterioles, as described by Dr Johnson, is, no doubt, a contributory, although only a secondary, cause of left ventricular hypertrophy. It is but the result of a reaction, or effort at self-adjustment of these vessels, which, subjected for some time to increased pressure from the left ventricle, undergo proportionate development, and acquire a corresponding power of resistance. This resistance, in turn, reacts upon the left ventricle, and determines still further hypertrophy of its walls.

Action and reaction, with alternate increase of muscularity of the ventricle and of the minute arteries, might thus go on indefinitely, were it not for failure of nutrition, and consequent tissue deterioration, in both. In the ventricle this assumes the character of fatty degeneration of the muscular fibre, and induces debility and dilatation; whilst in the arteries it consists in a similar change of the contractile fibre-cells, and atheroma of the middle coat, with consequent liability to aneurismal dilatation and rupture.

Bright found the heart enlarged, without pericarditis or valve-disease, in twenty-nine out of one hundred cases of albuminuria. In seventeen of these cases the left ventricle was the portion of the heart affected; and in the remaining twelve the part is not mentioned.* Whilst regarding the increased development of the left ventricle in albuminuria as the result of its abnormal func-

* *Guy's Hospital Reports*, vol. i., 1836.

tional activity, and the latter as due to the stimulating quality of the blood by retained excreta, he considered that this operated, not alone indirectly through the spasmodic resistance to the circulation which it induced in the small arteries, but also directly upon the heart itself.

Jaccoud* is disposed to accept, in part, the mechanical explanation of hypertrophy of the left heart in renal disease propounded by Traube; but, in many cases, he thinks with Bright, it may be due to the altered state of the blood.

Doctor Grainger Stewart gives the following as the proportions in which hypertrophy of the heart was present in the different forms of Bright's disease.† Of the "inflammatory form," it was present, and apparently referable to the disease of the kidneys exclusively, in the proportion of 40 per cent., the proportion increasing with the progress of the disease up to the last stage, when it amounted to 100 per cent. Of the cases fatal in the first stage, hypertrophy was present in the proportion of 12 per cent.; of those fatal in the second stage, in the proportion of 38 per cent.; and of those fatal in the third stage, or that of contraction, it existed in the proportion of 100 per cent.‡

In the "amyloid form," it existed only in the proportion of 4 per cent. of the cases; and in all these the disease of the kidneys was in its third, or atrophic stage.

Of cases of the "cirrhotic form," 46 per cent. exhibited enlargement of the heart, assignable to no other cause.

Quain states that the left ventricle was hypertrophied in twenty-one cases out of twenty-six of granular disease of the kidneys.§

The connexion between chronic renal disease and left ventricular hypertrophy, as cause and effect, may, therefore, be regarded as well established; and the progress of the latter affection, its consequences, and the collateral changes in other organs and tissues, fully ascertained.

* *Clinical Lectures on Medicine*, delivered at the Charité Hospital, Paris, 1867.

† *Bright's Diseases*, 1868 p. 41.

‡ *Opus citat.*, second edition, p. 90.

§ *Loco citat.*

Amongst the mechanical causes of hypertrophy of the heart, Roger mentions the pressure made upon it by a deformed thorax.* This is, no doubt, a not infrequent cause of hypertrophy with dilatation of the right side of the heart, as previously stated by Rokitansky, owing to the mechanical impediment usually presented to the circulation through the lungs; but the left side is not affected, except secondarily and remotely, and then by dilatation only.

Doctor Law maintains that, in the absence of patency of the foramen ovale and of an imperfect septum ventriculorum, hypertrophy of the right ventricle is scarcely ever met with. In the exceptional cases, hypertrophy of the right ventricle is, in his opinion, due to the stimulating action upon it of the arterial blood which finds entrance from the left side.† I have already expressed the opinion that hypertrophy of the right ventricle is very rare. The pathological associations in which it may occur are, no doubt, chiefly those mentioned by Drs. Law and Yeo (*vide* p 492); but others should be admitted; for example, the pressure of a tumor, aneurismal or cancerous, upon the trunk of the pulmonary artery.

There may, indeed, be hypertrophy of the right ventricle from emphysema of the lungs exclusively, as mentioned by Stokes,‡ and without its being indicated by precordial dulness, or by such only in a slight degree. I have, myself, exhibited before the Pathological Society a heart illustrative of the truth of this remark.

Doctor Clifford Allbutt§ would constitute mechanical diseases of the heart, I think unnecessarily, a distinct class. He states the form which they assume, and their relative frequency of occurrence to be the following:

1. Dilatation of right ventricle.
2. Dilatation of left ventricle.
3. Hypertrophy of left ventricle, or of both ventricles.
4. Chronic inflammation of aorta, and aortic valves.
5. Dilatation of aorta.

* *Journal of Practical Medicine and Surgery*, November, 1866.
† *Pathological Society*, April 14th, 1866.
‡ *Dublin Journal*, vol. ix , old series.
§ *St. George's Hospital Reports*, vol. v , 1870.

6. Incompetency of aortic valves.

7. Further compensatory hypertrophy of left ventricle.

8. Loss of compensatory power of left ventricle, with rapid failure, and often with mitral regurgitation.

These views are in some respects original; but I cannot coincide in the opinion of the author, that there is anything peculiar in the pathology of these cases, either as to the nature of the morbid changes, or the mode and order of their appearance.

Aneurism of the aorta has been, by many eminent authorities, regarded as a cause of hypertrophy of the left ventricle. Bertin has so regarded it;* so also have Rokitansky,† and Fuller;‡ and Lebert, Niemeyer, Oppolzer, and Bamberger, on the authority of Professor Axel Key of Stockholm.§ The last named eminent writer, however, declares that aortic aneurism has no such effect upon the heart, however rational it may seem to expect that it should. He has, on the contrary, met with examples of atrophy of the heart in connexion with aneurism of the arch of the aorta. When the valves are healthy, aneurism, in his judgment, exercises no influence upon the heart in regard to its size, form, or condition in other respects, and when hypertrophy or dilatation exists, it is due to an accompanying valvular lesion. The absence of hypertrophy he attempts to explain, by assuming an interrupted pulmonary circulation through pressure of the aneurism upon the pulmonary artery, its branches, or upon the lungs themselves. The tumor, moreover, by abstracting largely from the quantity of blood in actual circulation, tends to diminish rather than to increase the volume of the heart; and lastly, it favours this result, in his judgment, by impairing general nutrition, and inducing a state of anæmia.

But pressure of an aortic aneurism upon the pulmonary artery, or upon the lungs, of such a kind as to obstruct the circulation in them, is certainly an exceptional occurrence. Whilst, as long since shown by Dr. Stokes, aortic aneurism does *not* entail failure of nutrition, or anæmia, except where

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* *Traité des Maladies du Cœur*, p. 350.

† *Pathological Anatomy*, vol. i., p. 163.

‡ *Diseases of the Chest*, p. 659.

§ *Medical Times and Gazette*, June 4th, 1870.

it presses upon and obstructs the thoracic duct. Putting out of consideration, however, the soundness of his reasoning, the statistics furnished by Professor Axel Key are of great value as a contribution towards the settlement of this question. He has had, in all, seventeen cases of aneurism of the aorta; and all, with one exception, situated near the heart. Yet, in no single instance were the walls of the heart hypertrophied; in comparatively few were they even of normal thickness; and in all the remaining cases they were actually thinned, either without any, or with a very partial and localized dilatation of the left ventricle. Instead of hypertrophy, he avers that, taking into consideration the actual volume of the left ventricle, he has found atrophy of the walls of that chamber to exist in the majority of cases, even of large aneurism of the arch of the aorta. Long previously, however, as Axel Key has admitted, Dr. Stokes had declared that the connexion* of hypertrophy of the heart with aneurism of the aorta, was only accidental   He says: "In some instances, hypertrophy has preceded the aneurism, or there has been a deficient state of the aortic valves; but where the heart or its valves have not been previously engaged, there is no reason to believe that the existence of aneurism, in any portion of the aorta, throws additional labour on the heart, and hence we commonly find a healthy heart coexisting with a vast aneurism."† My own statistics are drawn from a total of twelve cases of aneurism of the intra-thoracic aorta, ten of which engaged the arch, and two the inferior portion of the vessel. Ten of the patients were males, and two females; and in two only out of the twelve, valvular disease existed, and this to a very trivial extent. Seven of the cases were fatal. The average duration of aneurism in these seven, as calculated from the most reliable evidence that could be obtained, having been eleven months and eleven days.

Of these seven, only two, aged thirty-three and fifty-six respectively, exhibited hypertrophy of the heart; and this but in a very slight degree, and affecting *equally* the right and left ventricle. Both these last mentioned cases presented extensive

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* *Ibid.*, June 11th and 18th, 1870.
† *Diseases of the Heart and Aorta*, 1854, p. 579.

and advanced atheroma of the aorta; the subjects of both were intemperate, and one of them was, moreover, a man of very laborious habits, a porter in the egg-market, and accustomed to lift and carry heavy hampers some distance upon his shoulders. The other, an architect by profession, was likewise of active habits, accustomed to long walks, and to the ascent of high scaffolding in his professional avocation. The aneurism in both engaged the arch of the aorta. In two other cases out of the seven, the heart was in a state of atrophy. The ages of these patients were thirty-four and twenty-eight respectively; both were intemperate, and in both the seat of aneurism was the descending portion of the aorta, below the arch. The aneurism in the latter of these (William Fay) was of enormous size, displacing the heart and liver, and descending into the abdomen. There was "slight" atheroma of the arch in one of these two, and in the other the vessel was apparently sound, except in the seat of the aneurism. In the three remaining cases of those which were fatal, the heart was normal as to size and capacity. The ages of the patients were, thirty-one, forty, and fifty-four years respectively. Two were temperate (one a female); the aorta was "highly" atheromatous in one; and no note as to the state of this vessel exists in connexion with the other two cases.

From the foregoing statistics I conclude that aneurism of the aorta, in any portion of its course, is alone not capable of giving rise to hypertrophy of the left ventricle; that it is quite as often associated with atrophy, as with hypertrophy of that chamber; that it is more frequently associated with a normal condition of the left ventricle, as to capacity and thickness of walls, than with either hypertrophy or atrophy of it, and, finally, when hypertrophy of either ventricle is actually found in association with aneurism of the aorta, it is, in the case of the left ventricle, either anterior in date to the aneurism, or, if cotemporaneous with it, the result of a different and coexisting lesion, generally valvular. The occurrence of hypertrophy of the right ventricle, in connexion with aneurism of the aorta, is nothing more than a coincidence, the result of obstruction in the pulmonary circulation; it is in no degree related, as Fuller surmises, to reflected obstruction in the aorta.

The singular combination of symptoms constituting Graves' or Basedow's disease, namely, persistent palpitation of the heart, enlargement and pulsation of the thyroid body, and projection of the eyes, has been by some authors regarded as a cause of hypertrophy of the heart. According to Stokes, the affection consists essentially in functional disturbance of the heart, which may be followed by organic change; namely, hypertrophy and dilatation of the left ventricle. He adduces two examples from the writings of Sir H. Marsh and Professor R. W. Smith of Dublin, in both of which death was the result of intercurrent disease; and in both there was hypertrophy and dilatation of the left ventricle; but both likewise presented valvular disease. In Marsh's case the auriculo-ventricular valves on the two sides of the heart were thickened on the margins, and the auricles were much dilated, showing that reflux upon them had been for some time in operation. In Professor Smith's case, hypertrophy and dilatation existed in a still higher degree, although the valve affected (the semilunar of the aorta) presented only the "slightest appearance" of structural change.* With coexistent valvular lesion, it must be conceded that the evidence afforded by these two cases is not conclusive in support of the alleged dependence of left ventricular hypertrophy upon the functional excitement of the heart in Graves' disease.

Trousseau regards the affection as "a neurosis which principally affects the heart and the supra-diaphragmatic arteries." He says, the heart may present temporary alterations, similar to those which are met with in pregnancy; and in rare cases the cardiac lesion is permanent when the neurosis has been of long duration. The temporary lesion of the heart in pregnancy, alluded to by Trousseau, is undoubtedly hypertrophy; hence, it would seem that in the rare instances adverted to by him, the permanent lesion was of that character. He has met with hypertrophy in Graves' disease unaccompanied by valve lesion; but, as he judiciously remarks, this condition may have preceded the advent of the disease in question.†

Graves himself considered the cardiac lesion, in the affection

* *The Diseases of the Heart and the Aorta*, p. 297.
† *Clinical Medicine*, Sydenham Society's edition, vol. i., p. 571-88.

which now bears his name, to be passive dilatation, temporary or permanent.

Niemeyer denies the occurrence of secondary dilatation with hypertrophy of the heart in this affection; but admits that when it is fatal, which is very rarely the case, weakness and gradual dilatation of the heart, followed by anasarca and œdema of the lungs, are the immediate causes of death.[*]

Professor Austin Flint has had under observation six examples of this affection, and in one only was there valve-disease. In the remaining five, when first examined, there was no enlargement of the heart; but in one of these enlargement was subsequently produced by long continued functional derangement. He regards the heart affection as the primary lesion, and enlargement of that organ as a usual consequence of the disease when chronic. He gives the case of a female, aged twenty-eight, in which, at first, no evidence of hypertrophy could be detected: after an interval of eight years, however, notable enlargement of the heart had been produced, as judged by displacement of the organ outwards, and increase of precordial dulness. She died a few months subsequently, and the heart was found to weigh twenty ounces; *but the kidneys were fibrous, and weighed fourteen ounces and a-half*[†] The words given in italics render this case valueless as evidence of a pathogenetic connexion between Graves' disease and hypertrophy of the heart.

In a future chapter I shall have occasion to discuss at length the pathology of this disease. Here it will suffice to observe, that I regard it as primarily and essentially a disease of debility, and belonging to the class of cardiac neuroses. Organic lesion of the heart, as due to this disease, is exceptional, and when present, and dependent upon it exclusively, I believe it consists invariably in dilatation with thinning of the walls of the heart. I have treated, since I have been an hospital physician, seven cases of the affection, and of all these I have made careful and repeated examination, and kept copious notes. There was no

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[*] *Text-Book of Practical Medicine*, translated by Humphreys and Hackley, vol. 1, p. 374.

[†] *The Diseases of the Heart*, third edition, 1870, p. 311-17.

death amongst them whilst under my observation, and therefore I have no *post mortem* evidence to adduce. But from physical exploration I am able to state, with certainty, that enlargement of the heart existed only in two out of the seven cases, and in one of these two there was a systolic murmur at the apex, leading to a well-grounded suspicion that structural disease of the mitral valve existed, and was the cause of hypertrophy. In the second of these cases, the disease had lasted eight years, and the physical evidence led to the diagnosis of dilatation, rather than hypertrophy of the heart.

Males are more frequently affected with hypertrophy of the heart than females, in the proportion of two to one, according to Walshe,* and it is more common in advanced age than in youth, according to the same authority. Vanderbyl found, out of forty examples of hypertrophy, thirty-four in males, and only six in females. The proportion of two to one, in favour of males, may be accepted as an average ; a difference which, no doubt, arises from the more laborious duties devolving upon the male sex in all civilized communities ; not from any inherent proclivity in sex, as such. As regards individuals, duties which impose more physical labour, and more exposure to extremes and rapid vicissitudes of temperature, entail proportionately greater liability to hypertrophy of the heart.

The rate of progress of hypertrophy is liable to great variety, according to its cause, the habits and age of the patient, and the comforts with which he is surrounded. Hope mentions a case which ended fatally within a year; and another with adherent pericardium and dilatation, in which death occurred in nine months from the date of first reported illness.†

Bertin mentions a case of simple hypertrophy, which he believed to have been developed in fifteen days (')

The renal form is that which soonest ends in death, because, when it occurs, formidable disease of another and a vital organ has already attained an advanced stage of progress.

Next in order are those acute forms of hypertrophy from rupture of the aortic or mitral valve. But in none of these cases

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* *Opus citat.*, p. 281.   † *Opus citat.*, p. 278.

is hypertrophy the sole, or even the chief cause of a fatal issue, although it may precipitate that event through pulmonary hæmorrhage, or by rupture of a diseased cerebral artery. It may also promote an unfavourable issue otherwise, by constituting a necessary step towards tissue-degeneration and dilatation of the heart, and failure of the circulation.

If the habits of the patient be dissipated, and thereby calculated to impair nutrition; if, at the same time, they be laborious, to the extent of overtaxing an impaired organ ; if the patient be of advanced age, and thereby more prone to degenerative change of tissue ; and, finally, if the wants of the body, as to food, rai-·ment, and external heat, be inadequately supplied, the progress of hypertrophy of the heart to a fatal issue will attain its maximum rapidity.

Doctor Gerald F. Yeo exhibited before the Pathological Society of Dublin,* a morbid specimen, illustrative of the dependence of hypertrophy of the right ventricle upon atheromatous degeneration of the pulmonary artery. A woman, aged thirty-two years, subject to congestion of the lungs, and who had been under observation for six years preceding, was admitted into the Whitworth Hospital for valvular disease of the heart, following acute rheumatism. The lungs exhibited several distinct foci of sanguineous infarction, and the branches of the pulmonary arteries leading to these were plugged with hard laminated fibrin. The pulmonary arteries, to their finest divisions, were dilated and inelastic, and their inner coat was marked with dull yellow patches of atheromatous degeneration. The auricles were dilated, and their appendices were filled with dry crumbling fibrin. The mitral orifice was contracted in an extreme degree, and the mitral valve greatly indurated. The aortic valve was thickened and insufficient. The left ventricle was hypertrophied, and the right ventricle dilated and greatly hypertrophied ; its walls were firm, and in some places three-fourths of an inch thick. The aorta was healthy. The tricuspid valve was thickened. Dr. Yeo remarked that engorgement of the lungs, though it may have aided, was of itself incompetent to produce this extreme hypertrophy of the right

* Meeting, December 14th, 1872.

ventricle, which, in his judgment, was caused by the morbid alteration in the pulmonary artery.

The *symptoms* and *signs* of hypertrophy must be considered separately, in reference to each of the different forms of the affection.

For the differential diagnosis, Corvisart relied mainly on the general condition of the patient, and the character of the pulse. In "active aneurism" (dilated hypertrophy), the patient is strong, and the pulse strong, hard, and vibrating, and there is visible pulsation of the carotids; whilst in the "passive" form (dilatation) there is general debility, and the pulse is feeble, soft, and weak.[*]

Morgagni also relied upon these symptoms as evidence of the dilated form; but Bertin held that they belonged not to this, but to the "simple" form of hypertrophy, of which he gives three examples (observations 34, 75, and 76) affecting the left ventricle, and remarks on the absence of lividity of face, capillary engorgement, œdema, and oppression; all of which, he adds, are present in hypertrophy with dilatation, and are due to the latter condition. Hypertrophy of the left ventricle tends to produce cerebral congestion and apoplexy, epistaxis, and congestive inflammation of the eyes; and hypertrophy of the right ventricle tends similarly to the production of pulmonary apoplexy. These forms of congestion he distinguishes as "active;" and apoplexy supervening on them he designates "*coup de sang.*"[†]

In "simple" hypertrophy Bertin says the sounds of the heart are more dull, remote, and prolonged than natural, but limited to the precordium; and in the "concentric" form the sounds are still more masked, and contrast with the force of the impulse.

In excentric hypertrophy, the sounds are louder, clearer, and more extensively diffused than in health, representing the *coup de marteau*, and are audible posteriorly.

Hypertrophy without complication, he declares, involves no alteration of rhythm, but it predisposes to palpitation on exertion; and when intermittence or great irregularity of the heart's action is observed in connexion with hypertrophy, it is due to other lesions with which the hypertrophy is associated, *e.g.*, narrowing of the orifices, or aneurism of the aorta.

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[*] *Opus citat.*, p. 130.   [†] *Opus citat.*, p. 283-9, and 350-1.

Respiration, according to Bertin, is not affected by uncomplicated hypertrophy, except when the latter is excessive, and presents a mechanical impediment to the expansion of the lungs; and in the concentric form, when the same embarrassment to respiration should exist as in the case of a contracted orifice. He dwells upon the fact, not previously recognized, that hypertrophy does not, of itself, impede the circulation, save in the concentric form; and that the effects hitherto attributed to it were, in reality, due to a preexisting organic lesion, which gave rise equally to the hypertrophy and to the symptoms ascribed to it.

Hypertrophy of the left ventricle is characterized by pulsation, strongest in the region of the left fifth and sixth costal cartilages; and hypertrophy of the right ventricle by pulsation of greatest force at the lower end of the sternum, and stronger to the right than to the left of that point. The latter is likewise distinguished by "active" hæmoptysis and jugular pulsation or "fluctuation," which, though regarded by Lancisi as indicative of right ventricular hypertrophy, was justly regarded by Bertin as symptomatic rather of dilatation of the ventricle and inadequacy of the tricuspid valve.

The combination of hypertrophy of both ventricles in different forms may be recognized by adverting to the place of greatest intensity, and to the character of the pulsations and sounds of the heart. Hypertrophy of the auricles is characterized, he says, by the muffled quality of the second sound.*

The authority of Bertin is so justly high, and his opinions so fully representative of the knowledge of his time in this department of clinical medicine, that I have deemed it desirable to state them thus at length, in order to shew in what modern teaching would correct and supplement his rules of diagnosis.

Intermittence and irregularity of the action of the heart, occurring in connexion with hypertrophy, should not be regarded, as justly stated by Bertin, as arising from hypertrophy as such. He was, however, wrong in regarding these symptoms as indicative of narrowing of any of the orifices of the heart, or of aneurism of the aorta. They indicate rather degeneration and dysæsthesia of the heart, and dilatation of the left ventricle. In enumerat-

* *Traité des Maladies du Cœur*, 1824, p. 350, *et suivantes.*

ing the topical signs of hypertrophy, Bertin omits all mention of the most valuable of them, namely, displacement of the point of apex-pulsation to the left.

The value of hæmoptysis, as a symptom of hypertrophy of the right ventricle, is certainly very equivocal; many other diseases of the heart are more likely to give rise to it, and notably constriction of the mitral opening. But, even were this not so, it would be impossible to distinguish active from passive hæmoptysis with sufficient precision, for the purpose of differential diagnosis.

The error of attributing the second sound of the heart to the contraction of the auricles, which was common to Bertin, Laennec, and Bouillaud, and led these eminent men and their disciples into many and grave mistakes in physical diagnosis, has been discussed in a previous chapter, and needs only to be mentioned here.

Bouillaud mentions as primary and essential symptoms of hypertrophy, augmentation of the force and extent of the pulsations, and increased loudness of *both* sounds of the heart; and extension of dulness in, and sometimes arching or prominence of the precordium.*

But exaggeration of the sounds is characteristic of the mixed form only where dilatation is in excess of hypertrophy, as he elsewhere admits. In the simple form, the sounds are dulled. Percussion-dulness is not necessarily increased, for example, where the anterior edges of the lungs are emphysematous, and overlie the pericardium; and prominence of the pericardium is not only possible in acute pericarditis with copious liquid effusion in the child, but infinitely more common than it is in connexion with hypertrophy at any period of life. He adopts, from his teacher Bertin, the error of attributing derangement of rhythm to stenosis of one or other of the orifices of the heart, and omits, as Bertin did, to mention the actual cause of irregularity, namely, degeneration of the muscular substance of the heart. Laennec states that, whilst moderate hypertrophy of the ventricles causes dulness of the first sound, and exaggerates the natural rhythm of the heart by prolongation of the long pause,

* *Traité Clinique,* tom. ii., p. 440.

extreme hypertrophy prolongs the systole, and either completely masks the first sound, or admits only a faint murmur like that of respiration. There is an obscure and distant movement which gradually increases, lifts the ear, and gives the sensation of a shock. The ventricular contractions run almost into one another, and there is scarcely any interval of repose, the second sound being curt, and almost or entirely lost.*

As symptoms of hypertrophy of the left ventricle, Laennec mentions a full and strong pulse; strong and visible impulse; diminution or absence of percussion-resonance in the precordium, and florid complexion; but adds, with truth, that none of the symptoms are constant. Laennec, like Bertin, omits all mention of displacement of the apex and extension of percussion-dulness to the left, as signs of hypertrophy of the left ventricle.

According to Hope, prominence of the precordium and "a double jogging impulse" are symptomatic of an hypertrophied heart, fixed by adhesion of the pericardium.† He truly remarks that the pulse in hypertrophy, complicated with "valvular and other lesions," is liable to many and great modifications, which represent rather the complications than the hypertrophy; but in left ventricular hypertrophy of the simple form, and free from complication, it is regular, strong, full, and tense; it swells gradually and with force, expands largely, and dwells under the finger, and in anæmic subjects it is accompanied with a thrill. In hypertrophy with dilatation, provided the former condition predominate, these characters of the pulse are still more decided; but when dilatation is in excess of hypertrophy the pulse is soft and compressible. In concentric hypertrophy the pulse is tense, but small; and where this condition exists in an extreme degree, it is weak and small.

He holds that dyspnœa, venous congestion of the lips, and passive hæmorrhage, may be caused by uncomplicated hypertrophy, through embarrassment of the capillary circulation; and that angina, although characteristic of no particular lesion of the heart, is often associated with hypertrophy.

The impulse of the heart is strong, heaving, and sustained, in

* *Traité d'Auscultation Médiate*, 1826, tom. ii., p. 411.
† *A Treatise on the Diseases of the Heart*, etc., p. 238.

proportion to the degree of hypertrophy and accompanying dila-tation; and in both the excentric and simple forms Hope notices, with especial emphasis, the existence of a "diastolic impulse," for the discovery and interpretation of which he justly claims credit.

In reference to this sign, he says: "It is occasioned by the diastole of the ventricles, during which action the heart sinks back from the walls of the chest, and this sinking back termi-nates in a jog, or shock, occasioned by the refilling of the ven-tricles, and constituting the diastolic impulse in question. It is stronger, *cœteris paribus*, in proportion as the heart is thicker and more capacious. Accordingly, I have found it strongest in hypertrophy with dilatation; but it may also be very consider-able in simple hypertrophy. In the healthy heart it is not per-ceptible; neither is it in dilatation without hypertrophy "[*]

Hope agrees with Laennec in thinking the ear, applied to the stethoscope, the most delicate medium for the appreciation of cardiac impulse. Jugular pulsation he regards as a sign of dilated hypertrophy of the right ventricle, maintaining that the double venous fluctuation is due, the lesser, to the wave of blood urged on by the contraction of the auricle towards the distended ventricle, and thence reflected upon the superior cava and its tributaries; and the greater, to the sudden and powerful contrac-tion of the ventricle upon the mass of blood filling its cavity, and the consequent repulsion upon the auricle, and through it upon the jugular veins, of a column of blood, equivalent in sectional area to the surface of the tricuspid valve thus suddenly and forcibly raised by the contraction of the ventricle.

The heart, he says, occupies a low position in hypertrophy, ex-cept when fixed by adhesion of the pericardium, and the sounds are modified according to the form of hypertrophy actually pre-sent. Thus, in simple hypertrophy the first sound is dull and prolonged in proportion to the degree of thickening of the walls, whilst the second sound is feeble, and the interval of repose is shortened because of the increased length of the first sound.

In concentric hypertrophy, he says, both sounds are propor-tionately weaker; and in the simple and concentric forms alike,

[*] *Opus citat.*, p. 271 2.

the first sound is scarcely audible at the upper part of the sternum and beneath the clavicles, whilst the second is distinctly heard. Where both ventricles are thus altered, the transmission of the sounds is restricted to the narrowest limits.

In hypertrophy with dilatation the sounds are loudest, and during palpitation they attain their utmost intensity. The first is abrupt at its commencement, and prolonged, whilst the second is simply intensified; both are extensively transmitted through the chest, and distinctly audible at all points of its surface, even behind, especially in children and thin persons.

Where dilatation predominates, the first sound, he says, is not so loud or prolonged as in the preceding variety, whilst the second is louder than natural, but otherwise unaltered.

Both the impulse and the sounds are diminished by general debility, induced by exhaustion from any cause; by acute and chronic affections of the lungs; and by approaching dissolution.

I have already expressed the opinion, that precordial prominence is not characteristic of hypertrophy, because proportionately more frequent in hydro-pericardium; and that elevation of the point of apex-beat, without any discoverable cause of excentric pressure on the heart, is indicative of adhesion of the pericardium exclusively. The "double jogging impulse" in no respect differs from the double impulse, systolic and diastolic, to be presently noticed.

Symptoms of venous engorgement are certainly not characteristic of uncomplicated hypertrophy in any of its varieties, quite the reverse; and dyspnœa is so only in a qualified sense, namely, when the hypertrophy is excessive, and incommodes the lungs by its mere bulk and pressure; or when it is induced by unwonted physical effort of any kind. The capillaries are not engorged as a consequence of uncomplicated hypertrophy. When they are in that condition in connexion with hypertrophy, the cause of the hypertrophy is also the cause of the capillary engorgement, e. g., valvular disease. In the early stages of hypertrophy the capillaries are not engorged, but in a state of active hyperæmia. When, however, in the latter stages, the heart begins to fail, passive congestion of the capillaries and veins ensues: a change due, not to hypertrophy, but to the atrophic and degenerative

changes of tissue which constitute its inevitable sequelæ. Then, for the first time, the pulse is noticed to be irregular, and dropsical effusion appears.   I regard as of no weight against this view the case of alleged concentric hypertrophy, causing dropsy and other consequences of capillary and venous congestion, adduced by Hope,* in the absence of positive evidence that the kidneys were not diseased.

The interpretation of the "diastolic impulse" given by Hope is, in my judgment, only partially correct.   Such impulse is present only where hypertrophy with dilatation exists ; never in simple hypertrophy.   His explanation of the phenomenon implies two coincident and essential conditions : a "sinking back " of the heart, and diastole of the ventricles.   I believe the latter constitutes the sole cause ; the former having no existence, and, therefore, no share in the production of diastolic impulse.

It is no less perceptible when the patient lies face downwards, and no "sinking back" can take place.   Further, Hope's theory implies that diastolic impulse takes place at the *end* of ventricular diastole, and coincides, therefore, with auricular systole.   But, however plausible this doctrine may seem, as introducing the element of active impulsion of blood from the auricle, it is opposed to observation, which shows that the diastolic impulse coincides with the second sound of the heart, and therefore with the *first* period of ventricular diastole.   My view is, that the hypertrophied, dilated, and dense walled ventricle, returning by recoil to a state of complete diastole, and becoming at the same moment suddenly and rapidly filled with blood by influx from the auricle, attains thereby dimensions in excess of the antero-posterior diameter of the mediastinum ; and partly by its diastolic recoil, and in part by the excentric pressure thus exercised upon its internal surface, it is brought suddenly into violent collision with the precordium, and becomes the cause of sensible impulse.   It is, I believe, pathognomonic of dilated hypertrophy of an unsoftened left ventricle.   With an exaggerated systolic impulse, this phenomenon constitutes the "double jogging impulse" previously mentioned, as indicative, according to Hope, of hypertrophy, with adherent pericardium ; but, as I

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* *London Medical Gazette,* September 5th, 1839.

think, erroneously so interpreted. Neither the explanation offered by Hope of jugular pulsation, nor his interpretation of that phenomenon, is, to my mind, satisfactory. I believe it to be due, as already stated, to actual reflux from the right ventricle; and that it is, therefore, pathognomonic of tricuspid regurgitation. There are noticeable in the external jugular veins, two distinct movements in such cases: one, a single reflux wave of considerable magnitude, coincident with the systole of the ventricles and the carotid pulse, and all but abolished by a full inspiration; and the other, a series of ripples, likewise reflux, but in no degree influenced by respiration, alternating with the larger fluctuations, and occupying the whole interval between them. Both these movements may be promptly arrested by gentle pressure upon the vein, immediately above the clavicle. The former I believe to be due to reflux from the ventricle during its systole; and the latter, to reflux from the auricle during the series of feeble undulatory contractile movements performed by it previous to its final systole.

The systole of the auricle, properly so called, which immediately precedes the systole of the ventricle, is not productive of distinct venous reflux. The tricuspid orifice being thrown open at the moment of auricular systole, the blood passes by the larger opening into the ventricle; it is urged by the onward contraction of the auricle, and its course is favoured by gravitation, more freely than by the smaller aperture and against gravity, back upon the cava and jugular veins.

It is possible, however, that a reflux jugular wave may be actually produced by right auricular systole; but it would be necessarily merged in that of ventricular systole which immediately follows, and therefore not separately recognizable.

It may be and has been urged against this explanation of venous pulse in the neck, that it necessarily implies the concurrent development of tricuspid reflux murmur. But where the distention of the ventricle is such as to render the tricuspid valve incompetent, its power of contraction is manifestly insufficient to educe a murmur. Hence, the extreme rarity of tricuspid regurgitant murmur; so rare, indeed, is it, that I have never heard it except in cases, very few in number, in which incom-

petency was the result of valvular lesion without distention of the ventricle.

In speaking of an abbreviated interval of repose as a collateral sign of hypertrophy, Hope does not make it clear to which of the two intervals he refers. The first interval is shorter; but the second, or that following the second sound and preceding the first, is longer than natural, as stated by Laennec, because the prolongation of the first sound is effected by *suffix*, not by prefix; ventricular contraction not anticipating, but *transgressing*, its normal period.

I have not met with an example of the double systolic impulse which Walshe notices as a sign of excentric hypertrophy.* I have occasionally observed, in the early stages of retrograde metamorphosis of an hypertrophied heart, an abortive pulsation or tick, not represented in the radial pulse, to immediately succeed the normal impulse at the precordium. But this is symptomatic, not of hypertrophy itself, but of the retrogressive changes consecutive to that condition.

Neither can I, in view of the negative and positive signs, admit with that eminent observer, the possibility of mistaking excentric hypertrophy in a narrow chested subject, for pleural effusion.

In extreme cases of dilated hypertrophy of long duration, and also in early youth, Walshe has noticed bulging of the precordium, divarication of the ribs on the left side from the second to the eighth, and percussion-dulness to the same extent; the head of the observer, the body of the patient, and even the bed being visibly shaken whilst the stethoscopic examination was being conducted. This, I have likewise witnessed, but only in childhood (case T. D.). I cannot agree with him in the opinion, that reduplication of the sounds possesses no special significance; but I accept his view of its dependence, in regard to *both* sounds, on inequality of strength in the two ventricles.† I first sound, as already stated (p. hypertrophy with dilatation; or de- on, and that it depends upon

he Heart and Great Vessels, third edition,

disturbance of synchronism between the systole of the ventricle, and the closure of the corresponding auriculo-ventricular valve.

Reduplication of the second sound is caused by unequal repletion of the two ventricles, and consequent derangement of synchronism in their evacuation, and in the consecutive reaction of the aorta and pulmonary artery; the closure of the semilunar valves being postponed on the side of greatest ventricular distention.

The doctrine that hypertrophy is capable, *per se*, of inducing murmur at the aortic or mitral orifice by the mere force of contraction to which it may give rise, is quite unsupported by the result of my observation. I have repeatedly, however, encountered such a phenomenon; but at the mitral orifice only, in hearts, whether hypertrophied or not, dynamically weakened by softening of the ventricular walls.* In no single instance have I met with aortic systolic murmur not traceable to some definite organic lesion of an obstructive kind, either at the orifice or in the root of the aorta, which was not manifestly of hæmic origin. I cannot, therefore, believe in the alleged occurrence of aortic systolic murmur from derangement of proportion between a dilated ventricle and its orifice of exit; or from derangement of the line of axis from the orifice of the ventricle to the aorta, till conclusive evidence be adduced of its actual occurrence.

Amongst the general symptoms of hypertrophy, Walshe mentions paroxysms of dyspnœa in case of much dilatation, valvular obstruction, or pulmonary disease. Dyspnœa, occurring in paroxysms in connexion with hypertrophy, I should regard as evidence of dilatation and rigidity of the aorta.

Particular tinting of the face, frequent headache, rushing noises in the ears, drowsiness after meals, startings in sleep, and epistaxis, although collectively or singly often present in hypertrophy, are quite as often absent; hence they can be regarded only as *presumptive* symptoms of that condition.

Enlargement of the thyroid body is assuredly not a consequence, and therefore not a symptom of hypertrophy. It is commonly associated with excitement of the heart, which may or may not be followed by hypertrophy; but in relation to both it stands as cause, not as effect.

* See Chapter III., p. 281.

In connective tissue hypertrophy, Quain states that the impulse is strong and heaving, and the first sound dull; and in that due to accumulation of fat, which he admits as a special form, without adequate reason in my judgment, the sounds are sharp.*

In the latter condition the sounds are, no doubt, in most cases sharp at the base, owing to the usually concomitant change of atheroma of the aorta. But the apex-pulsation is feeble, and the apex-sounds faint, and of low pitch, except in the advanced stage of concomitant degeneration of the muscular fibres, when the sounds, though faint, may be likewise sharp and brief.

The symptoms and the signs† of hypertrophy may be divided into those which are *presumptive* or *equivocal*, and those which are *positive*; and each member of these two groups must be considered separately in relation to the different forms of hypertrophy specified at page 462.

EQUIVOCAL.

Symptoms.	*Signs.*
Florid or congested face.	Extension of precordial dulness.
Liability to headache in recumbent posture.	Masking of cardiac sounds.
,, to rushing noise in the head on stooping, or after exertion.	Extensive diffusion of cardiac sounds.
,, to violent palpitation after exertion.	
,, to epistaxis.	
,, to vertigo	
Habitual shortness of breath on exertion.	
Throbbing of the carotid arteries.	

POSITIVE.

Large, full, and strong pulse.	Displacement of the apex to the left, in the absence of excentric pressure.
Visible heaving of the precordium.	Extension of area of apex-pulsation.
	Heaving precordial impulse, perceived by the application of the hand.
	Extensive diffusion of impulse.
	Prolonged, dull, and distant first sound.
	Reduplication of first sound.
	Double impulse.
	Sphygmographic tracing of hypertrophy.

* Lectures, *loco citat.*

† For convenience sake I use the word "sign," as contradistinguished from symptom, throughout this work, as indicating sensory evidence discoverable by auscultation, percussion, or palpation.

It is unnecessary to discuss here the equivocal symptoms and signs, which have but a casual connexion with hypertrophy; each of which may be absent where hypertrophy exists, or may be present in its absence, and due to other causes.

Of the positive symptoms of hypertrophy, the large, full, strong, and sustained pulse is amongst the most valuable, it is distinguished by the *combination* of these qualities from the pulse of all other morbid states whatever, and is, therefore, pathognomonic of left ventricular hypertrophy. It is liable to great variation as to firmness, according to the state of the walls of the artery examined; but, it is always of high tension, except in the stage of retrogressive change of tissue.

The absence of pulse of this character would not be of equal value as negative evidence in regard to the heart generally, because the right ventricle alone may be hypertrophied, and yet the pulse may present characters the opposite of those just mentioned.

In simple hypertrophy of the left ventricle, the pulse is less full and sustained, but no less strong, than in the dilated form. The difference, therefore, is only of degree, as between the simple and dilated forms of the affection on the left side of the heart.

Hypertrophy of the right ventricle is nearly always of the dilated form, and the radial pulse exhibits characters derived from an obstructed pulmonary circulation, viz., those of smallness, weakness, and occasionally even of intermittence or irregularity.

Visible heaving of the precordium is no less significant of hypertrophy, in the assumed absence of aneurism or other tumor behind the heart. When the left ventricle alone is affected, the movement will be perceived towards or beyond the line of the left nipple; and where the right alone is engaged, it will appear, but less distinctly, in the inferior sternal region, and to a variable distance beyond the right edge of that bone. If both ventricles be in the state of hypertrophy, the movement of impulse will be visible over the entire extent of the precordium, but most distinctly at the point of apex-pulsation; and in the dilated form it will be of an undulatory character.

As a single sign, displacement of the apex to the left of its normal position might be due equally to hypertrophy and dila-

tation of the left ventricle; and the same may be said of enlargement of the area of apex-pulsation. But, associated with heaving impulse at the left precordium perceptible to the hand, these signs, single or united, become pathognomonic of left ventricular hypertrophy. The degree of displacement of the apex to the left, and of extension of its area, will, in the association just mentioned, measure with approximate accuracy the enlargement of the left ventricle; and its downward and leftward displacement, the expansion and elongation of the organ so characteristic of hypertrophy from incompetence of the aortic valve. In determining the extent of apex-pulsation I make use of my index and middle fingers as a pair of callipers, and with sufficiently accurate results.

Heaving precordial impulse is no less distinctive of hypertrophy; it is of the nature of a prolonged swell or upheaving of the chest-wall, by the shock and gradual uplifting of a powerful solid body immediately behind it. In simple hypertrophy the shock is more abrupt and localized, and the lifting of the chest-wall less decided than in the dilated form. In excentric hypertrophy of extreme degree, the shock of cardiac impulse is so powerful as visibly to shake the body of the patient, and the head of the auscultator, as correctly stated by Hope. I have seen it communicated to an enlarged liver, and I have heard gastric tinkle, corresponding to the systole of the ventricles, caused by it. It is, moreover, extensively diffused over the front of the chest, being perceptible to the hand far beyond the normal limits of the precordium.

The first sound of the heart is strong, but dull, prolonged, and distant in all forms of hypertrophy. In simple hypertrophy the dull and distant characters of the sound predominate; and in the dilated form, prolongation and strength, with greater clearness, are the distinguishing qualities. The second sound is less distinctly audible at the apex in both forms than it is in the healthy heart; louder, however, in dilated than in simple hypertrophy. At the base, the second sound is usually distinct and normal; but in many cases of excentric hypertrophy it is morbidly loud, sharp, and clear, for some distance in the line of the ascending aorta. This is, in some degree, due to the force of im-

pulsion, and usually also to antecedent or consecutive atheroma of the coats of that vessel.

Both sounds are extensively transmitted in dilated hypertrophy, especially in young and thin subjects. In such I have heard them at all points of the chest, even posteriorly. A solidified lung would seem to intensify, by transmission, the sounds of the heart in these cases.

Reduplication of the first sound is so intimately associated with simple or excentric hypertrophy, that if carefully sought, it will.be found in every such case, anterior to consecutive deterioration of tissue. It consists, as already stated, in resolution of the first sound into its constituent elements of impulse and valve-tension; and is due to the abolition of simultaneousness of these events, and the interposition of an appreciable interval of time between the impulse by which the systole of the ventricles is inaugurated, and the tension of the auriculo-ventricular valves, which occurs at its acme.

The suddenness and energy with which ventricular contraction is initiated in simple hypertrophy, and the ordinary rate at which its acme is attained, explain at once the early occurrence of impulse, and the apparent postponement of valve-sound; whilst, in excentric hypertrophy, the derangement is due to postponement of valve-tension, arising from the protracted process of ventricular evacuation. In both, the exalted contractile force acquired through increased parietal development, accounts for the emphasis with which the two elements of the first sound are pronounced. The absence, in a simple dilated ventricle, of contractile power adequate to the production of audible impulse, may account for the absence in such cases of double first sound. Hence, likewise, double first sound has rarely, if ever, its source in the right ventricle; for, even when hypertrophy is associated with dilatation of this chamber, an event of very rare occurrence,* dilatation is so much in excess that audible impulse is scarcely developed, and inadequacy of the tricuspid valve is a

* Bertin has, however, reported a case (88) in which the walls of the right ventricle were from eleven to sixteen lines thick. Bouillaud gives one (76) in which the thickness was eight to ten lines, and a second (77) in which it was twelve lines; and Hope gives an example of it, measuring six to seven lines, in a girl nine years old.

necessary concomitant ; hence, absence of valve-click, and there-fore, of double first sound.

I have already (p. 499) stated my opinion as to the cause and diagnostic significance of the "double jogging impulse" of Hope. It will suffice here to repeat, that I regard it as identical with double impulse of systolic and diastolic rhythm, as described by that author ; and as evidence, not of simple hypertrophy with or without adhesion of the pericardium, but of hypertrophy with dilatation of the left ventricle. Dilatation of the right ventricle, as just stated, is rarely associated with hypertrophy of its walls. Hence, likewise, double impulse rarely has its origin in this chamber. I am not personally cognizant of a single example of the kind.

A sphygmographic tracing would, of course, afford conclusive evidence of hypertrophy of the left ventricle.

Hypertrophy of the right ventricle must be judged of by other evidence. The condition usually met with and designated hy-pertrophy, is that of dilatation of its cavity with normal thick-ness of its walls.

Veritable hypertrophy with dilatation of this chamber is, how-ever, of occasional occurrence. It has been met with in associ-ation with long continued mitral obstruction ; with pulmonary emphysema ; and with primary hypertrophy of the left ventricle from over physical exertion, and subsequently complicated with chronic bronchitis, in well nourished but gouty subjects.

General hypertrophy of the right ventricle may likewise be witnessed in connexion with the exceedingly rare lesion of ob-struction at the orifice of the pulmonary artery, without perfora-tion of the ventricular septum or patency of the foramen ovale. Where direct communication between the right and left side of the heart exists, by imperfection of the septa, the right ventricle may, according to Dr. Law, undergo hypertrophy from the circu-lation of arterial blood through its cavity.

Doctor Sutton holds that in this condition of the right ven-tricle, systolic *bruit de soufflet* may be developed at the tricuspid orifice by incompetency, without structural disease of the tri-cuspid valve.* It is, he says, characterized by being audible at

* *London Hospital Reports*, vol iv.

the ensiform cartilage, or a short distance to the left of the sternum at the level of the fifth and sixth costal cartilages, but *not* at the left apex or left scapula. He gives the details of eight cases in which he had personal knowledge of its existence, and in which he examined the body after death. The tricuspid orifice was dilated in all these cases; but, in several of them, likewise, the edges of the valves were rough. In a ninth example given, tricuspid reflux murmur existed, although, as proved by examination of the body, the right ventricle was simply dilated, but the valves were uneven, and manifestly incompetent. I have already stated that, even where veritable tricuspid reflux was proved to exist by jugular venous pulsation, in cases of distention of the right chambers of the heart with or without hypertrophy of the right ventricle, I have never heard tricuspid regurgitant murmur. I have, in a few cases, heard it where the right chambers were not engorged; but the valves had been rendered incompetent by roughening of their edges. I venture to suggest, and the facts as furnished by Dr. Sutton do not absolutely forbid this view, that, in those of his cases in which a rough state of the tricuspid valves existed, regurgitation and murmur would be thereby adequately explained; whilst in the others, the murmur may not have been adynamic and mitral; a murmur which is rarely audible in the left axilla, and never at the left scapula. Addison, as stated by Dr. Sutton, did not believe in tricuspid regurgitant murmur, and Hope and Walshe regard it as very rare.

The signs by which this condition of the right ventricle is manifested are few; and, with one exception, indifferently pronounced. The exception alluded to is the existence of strong impulse at the lower sternum and ensiform cartilage, in the absence of pulmonary emphysema, or other cause of displacement of the heart to this locality. The impulse in question is diffused, and of moderate strength rather than heaving; apex-pulsation is not perceptible, unless the left ventricle be also in a state of hypertrophy, because the apex is thrown backwards by the increased development of the body of the right ventricle.

Pronounced, and amply sufficient for the purpose of diagnosis, as are the signs of hypertrophy discussed in the last pages, the

evidence of its existence is not usually limited to these signs.
There are present, in addition, in the majority of cases, the signs
of valvular lesion, obstructive or regurgitant, which must lead,
and within a brief period, to hypertrophy of the corresponding
ventricle. The existence of a well pronounced systolic murmur
at the right base of the heart, traceable through the arch of the
aorta and into the carotids, with or without diastolic murmur
in the same situation, should suggest the coexistence of left
ventricular hypertrophy. A single and diastolic murmur here
would have a like significance. When aortic diastolic murmur
exists, the apex of the heart will be found to have undergone
displacement downwards as well as towards the left. Systolic
murmur having origin in the aorta, above its orifice, and dis-
tinguishable from obstructive lesion at the mouth of the vessel
by the higher level of its point of greatest intensity, by its non-
transmission into the cervical arteries, and by the coexistence of
rigidity and tortuosity of the superficial arteries, will be likewise
found associated with the positive signs of hypertrophy of the
left ventricle.

Mitral regurgitation also leads inevitably to hypertrophy of
the left ventricle. Therefore, when apex systolic murmur exists,
the distinctive signs of hypertrophy should be looked for. Not
so with mitral obstruction. In connexion with this lesion, if
unassociated with mitral inadequacy, or other cause of hyper-
trophy, the left ventricle is either strictly normal in all respects,
or somewhat reduced in capacity, but never thickened in its walls.

The existence, therefore, of simple presystolic murmur at the
apex, would be *primâ facie* evidence of the non-existence of
hypertrophy of the left ventricle; and the absence of the posi-
tive signs of that condition, already mentioned, would confirm
the negative diagnosis.

The *pathology* of hypertrophy is scarcely on a level with our
knowledge of its diagnosis.

Senac, Morgagni, and Corvisart, laid down the principle that
the heart became hypertrophied, like the external muscles, under
the stimulus of increased work.

Corvisart distinguishes the induration of "active aneurism"
from that arising from thickening of the connective tissue of the

heart,[*] and gives an example (Observation 27) of the latter kind, in which the pulse was small, hard, and slightly irregular, whilst the heart's action was strong and tumultuous; thus representing the want of harmony between the pulse and the heart, usually regarded as characteristic of aortic obstruction. At the necropsy, all the chambers of the heart were found dilated, rigid, and resonant, and the aortic orifice contracted.

Quain has recently[†] given a minute description of this form of hypertrophy, in the recognition of which he was, however, long anticipated by Corvisart, as above shown.

Corvisart also supplies an example of cartilaginous alteration of the heart (Observation 28), in which the symptoms were identical with those in the former case, and the pathological change, as he avers, only more advanced. [‡] Bouillaud gives several such.[§]

A condition of the heart corresponding to the description of fibroid cartilaginous change above given, must be extremely rare at the present time. I have not met with a single example of the kind, and I incline to think that its rarity has some relation to the less heroic treatment of hypertrophy now practised.

Rindfleisch attributes fibroid transformation to diffuse parenchymatous inflammation of the muscular substance of the heart; of which he gives an example in a syphilitic subject.[||] The heart was rigid, somewhat violet in colour, and in section iridescent. The cut edges were nearly translucent; the consistence that of caoutchouc, but unyielding, so that the fibres would tear before stretching. There were numerous ecchymoses beneath the endo- and pericardium, whilst the blood-vessels of the heart were empty. The interior of the muscular fibres was occupied by fine granules, which were accumulated in fusiform heaps around the nuclei; the fibres exhibited cross rents, the result of mechanical tearing.

The " fibroid patches" of the same writer are likewise regarded by him as of inflammatory origin, and engaging the substance of the heart by extension from the endocardium. The description

* *Opus citat.*, p. 146.
† Lumleian Lectures, 1872.
‡ *Opus citat.*
§ *Ibid.*, p. 152.
|| *Manual of Pathological Histology*, Sydenham Society's edition, vol. i., p. 275.

just given corresponds to that of "fibroid transformation" of the heart published by Ormerod many years ago ;* but it does not appear that hypertrophy existed in any of these cases.

Sir W. Jenner has shown† that chronic congestion of the heart may lead to "induration," "toughening," and thickening of its walls by interstitial deposit of lymph, and ultimately to permanent dilatation. The walls so affected, though thin, do not collapse on section. The tissue is either dark, or pale, close grained and leather-like, according to the stage of induration attained.

Under the microscope the striæ are seen to be imperfectly marked in distinct foci ; the fibres firmly cohere ; and between and within them protein granules are visible. Droplets of olein, and fibres in a state of veritable fatty degeneration, are likewise seen in a few places, and when such centres have multiplied and coalesced, softening of a previously indurated heart is the result.

Congestion of the heart, to lead to these results, must be gradually developed and long continued, but intermittent ; and the heart must have been, at the same time, actively functioning. Six cases are given by Sir. W Jenner, in all of which the patients had suffered from chronic but intermittent congestion of the thoracic and abdominal viscera; and after death the heart, liver, and kidneys were found to have undergone the changes above described. It is noteworthy that in three of these cases the mitral orifice was much narrowed.

In the further progress of fibroid change of the heart its substance may become rigid and resonant. Corvisart recorded an example of this kind, in which the heart, when struck, resounded like horn. Laennec met with a similar example. Burns‡ saw the ventricles converted into bone, like the vault of the skull. And Broussais gives two cases in which the heart was as firm as a cocoa-nut shell.

In reference to the prevalent opinion that in hypertrophy the muscular fibres have become actually thickened, Rindfleisch

* *British Medical Journal*, 1863.
† *Medico-Chirurgical Transactions*, vol. xliii , 1860.
‡ *The Diseases of the Heart*, p. 131.

observes that he has failed to discover any such change; and he intimates his opinion that hypertrophy is due to excessive cleavage of the fibres, by tension of their lateral branches during contraction.

I am not in a position to offer a positive opinion upon this doctrine. It seems to me doubtful that such a condition of the fibres could exist without lessening the density of the heart; yet, as is well known, the hypertrophied heart, previously to retrogressive change, is actually more dense than the healthy organ.

The alleged *consequences* of hypertrophy of the heart, respecting some of which well grounded doubts may be entertained, are the following:

Dyspnœa.
Dropsy.
Dilatation of the aorta.
Apoplexy, cerebral and pulmonary.
Fatty degeneration.
Gangrene.

That dyspnœa is not a necessary consequence of hypertrophy of the heart, is shown by its absence in pedestrians, pugilists, sledgers, oarsmen, etc., whose habits or occupation require the greatest and most protracted muscular efforts, and the highest breathing capacity. The compatibility of hypertrophy of the heart with great respiratory capacity, is likewise exemplified in the racehorse and greyhound. The heart of the celebrated greyhound, " Master Magrath," weighed 9·57 ounces avoirdupoise ;* and as the animal had won its last prize, the Waterloo Cup, only ten months previous to his death, and only two months before his death he beat, with facility, a kennel-companion in a trial coursing match; and as, in the absence of valvular, renal, and arterial disease, no cause of hypertrophy could be assigned, except the great and long sustained muscular efforts to which the dog had been accustomed, the conclusion seems inevitable, that it had coursed for and won in at least its last great prize, with a heart greatly in excess of the normal dimensions of that organ.†

* Professor Haughton, in *British Medical Journal*, January 20th, 1872.

† The weight of the dog's body in coursing condition was 54 lbs. ; that of the heart after death was 9·57 ounces avoirdupoise. The heart was, therefore, threefold in excess of the normal proportion of heart-weight to body-weight.

In the ordinary course of events, however, a period will certainly arise, if death do not intervene, when, owing to retrogressive or degenerative changes of its muscular tissue, the heart will fail as the circulating centre ; and vascular congestion and dyspnœa will inevitably follow   But, in all such cases, hypertrophy is a predisposing, not an immediate, cause of dyspnœa. The occasional paroxysms of cardiac dyspnœa which Corvisart witnessed in hypertrophy, and which he conjectured to have been due to " temporary fulness of the cerebral vessels, or those of the lungs,"* were manifestly of different origin. Paroxysmal dyspnœa of this character, as I hope to show in a future chapter, arises from dilatation of the aorta : a condition by no means infrequent in connexion with hypertrophy of the left ventricle, as the result of either concomitant or consecutive disease of the coats of that vessel. It is conceivable that a heart which had attained extraordinary dimensions, might, by its mere bulk, prevent the full expansion of the lungs, and so diminish respiratory capacity.

Dropsy, when consecutive to hypertrophy of the heart, is always due to adynamia or congestion from secondary dilatation, or to fatty change. Dropsy of this kind should, however, be carefully discriminated from renal anasarca associated with hypertrophy of the heart consecutive to renal disease ; it may be readily distinguished from the latter by the appearance, simultaneously with the dropsy, of venous and capillary engorgement, together with the physical signs of failure of the heart ; whereas, in the renal form, the dropsical effusion long precedes venous congestion and irregularity of the heart and radial pulse. Where fatty disease of the heart, primary or secondary, or of the kidneys, is the cause of anasarca, I have noticed that the scrotum and penis are swollen at a very early period of the disease.   I cannot, however, venture to explain the connexion.

Dilatation of the aorta is a direct result of dilated hypertrophy of the left ventricle, and may certainly occur without being preceded by disease of the coats of that vessel.   I have met with many examples confirmatory of this statement.   It would seem

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* *The Diseases of the Heart*, Hebb's edition, p. 164.

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as if the enlarged and powerful ventricle had operated injuriously upon the aorta, partly by its intrinsic force of contraction, and partly by the large mass of blood, out of proportion to the normal calibre of the vessel, projected into it at each act of systole. In consequence of this repeated surcharge and over distention, the walls of the aorta soon lose their elasticity, and the vessel remains permanently enlarged.* The usual changes of tissue consecutive to malnutrition are quickly set up in the coats of the aorta, after dilatation has been established. Over-distention of the vessel causes increased density of its walls; and from this follow, obstruction of the *vasa vasorum*, paresis of the trophic nerves, failure of capillary circulation, and arrest of nutrition in the walls of the aorta. Retrograde metamorphosis and atheromatous change are the results. Thus, whilst atheromatous degeneration of the arterial coats is usually primary and general, and constitutes a *cause* of hypertrophy of the left ventricle, in this instance it is secondary and local, and one of the *effects* of hypertrophy. But dilatation of the aorta may, and frequently does, lead to the further result of incompetency of the aortic valve, by simple derangement of proportion between it and the mouth of the vessel. In such case, two new factors of hypertrophy of the left ventricle are introduced; namely, atheroma of the coats of the aorta, and inadequacy of its valves. The progress of hypertrophy is proportionately accelerated, till such time, usually not distant, as retrogressive softening of its walls shall have been set up.

Dilatation, by yielding of the softened and enfeebled walls under the excentric pressure of the contained blood, now takes the place of hypertrophy, and vascular engorgement of the lungs, distention and dilatation of the right side of the heart, general venous congestion, and anasarca, follow in rapid succession.

In the progress of such a case, therefore, the occurrence of diastolic murmur at the right base would mark an era of great importance, as that of the " turn " of the case, after which general and rapid declension might be predicted.

* On the subject of the loss of elasticity in yellow fibrous tissue, under long continued or oft repeated tension, the reader is referred to a paper by the author in *The Dublin Quarterly Journal of Medicine,* 1861.

Cruveillier is likewise of opinion that hypertrophy of the left ventricle may cause dilatation and calcification, and even aneurism of the aorta.[*]

Amongst the alleged consequences of hypertrophy of the heart are *cerebral* and *pulmonary apoplexy.*

Corvisart, whilst theoretically admitting the occurrence of apoplexy by rupture of the cerebral arteries, either directly from the force of contraction of the left ventricle or indirectly from venous obstruction and reflux upon the arteries of the brain, declares that he cannot recall an instance in which "apoplexy was the evident consequence of a disease of the heart."[†] Cerebral venous congestion, however, he has repeatedly witnessed.

Bertin, as already stated, regarded hypertrophy of the ventricles of the heart as the immediate cause of active congestion and apoplexy of the brain and lungs.[‡]

Rokitansky admits an intimate connexion between simple and excentric hypertrophy of the left ventricle, and cerebral apoplexy; but he regards a friable state of the arteries of the brain as a necessary concomitant condition.[§] He denies the alleged causal relationship of right ventricular hypertrophy to pulmonary apoplexy.

Hope and Bouillaud regard the connexion between hypertrophy of the left ventricle and cerebral apoplexy, as direct and intimate; but, with Morgagni, they both considered it probable that in all such cases, disease of the arteries of the brain likewise exists. Hope, however, considers hypertrophy the principal factor. But out of four fatal cases of apoplexy under the age of forty years given by him,[||] not one presented disease of the heart; whilst, after that age, disease of the heart existed in twenty-five out of thirty-five fatal cases.

Andral is likewise a believer in the direct connexion of left ventricular hypertrophy with cerebral hæmorrhage; but, on the other hand, Fuller regards it as due entirely to the frequent

[*] *Anat. Pathologique*, l. xvii., pl. iv., figs. 1 and 2.
[†] *Opus citat*, p 164.
[‡] *Maladies du Cœur*, p. 351.
[§] *Pathological Anatomy*, vol. i., p. 169.
[||] *Opus citat.*, p. 256.

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association of atheromatous change in the arteries, with hypertrophy of the left ventricle.*

In regard to the alleged dependence of pulmonary apoplexy upon hypertrophy of the right ventricle, Rokitansky, as already shown, opposes this doctrine. Hasse and Stokes, on the other hand, uphold it. I believe, however, that derangement of the natural adjustment subsisting between the propulsive force of either ventricle, and the power of resistance of the corresponding arteries, can lead to rupture of the latter only by becoming the cause of disease of the arterial coats, as previously explained (p. 514); and that the occurrence of apoplexy by extravasation, in connexion with ventricular hypertrophy, implies unsoundness of the vascular walls. In the brain the arteries are exposed to a further but extrinsic source of weakness, from softening of the cerebral substance by which, in the normal state, they are supported, and, thereby, enabled to resist high pressure.

The connexion would seem to stand thus. Arterial disease is more common after forty; and the cerebral arteries, being destitute of a *tunica adventitia*, and inadequately supported by brain substance, readily give way when diseased, especially under the impulse of an hypertrophied left ventricle. But hypertrophy of the left ventricle is usually associated with arterial degeneration, either as the cause, or as a concomitant change dependent upon the same morbific agency; *e.g.*, chronic renal disease. Hence, apoplexy would seem to be the product of two concurrent causes; the one predisposing, viz., cerebral arterial disease, and the other immediate, left ventricular hypertrophy. Arterial disease is quite as often the antecedent condition and the cause of hypertrophy, as it is posterior in time, and dependent, however remotely, upon hypertrophy. If hypertrophy in the simple form be more likely than any other to be followed by apoplexy, as Hope surmises, it is because this is the form in which hypertrophy is oftenest found in conjunction with chronic renal disease in its early stages, when the twofold condition of strong propulsive power in the left ventricle, and atrophic changes in the arterial coats, exists.

Doctor Walshe † discusses this question with great acumen,

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* *Diseases of the Chest*, 1862, p. 579.
† *The Diseases of the Heart*, third edition, p. 290.

and arrives at the conclusion that the percentage of cerebral apoplexy, in those affected with hypertrophy of the left ventricle, is but slightly in excess of its ratio where no cardiac disease exists. If the further consideration, that hypertrophy in the great majority of cases is a condition of compensation, be allowed its legitimate weight, and that, with the two exceptions of renal hypertrophy, and hypertrophy consecutive to primary arterial disease, the obstruction to which it owes its origin is located on the cardiac side of the cerebral arteries, the conclusion will seem inevitable, that in hypertrophy of these two forms only, can cerebral apoplexy be regarded as even remotely dependent upon increased propulsive power in the left ventricle.

Whilst denying the deleterious influence of left ventricular hypertrophy on the cerebral circulation, Dr. Walshe is disposed to regard tricuspid inadequacy as a direct cause of cerebral venous congestion. But apoplexy from this cause is exceedingly rare.

Pulmonary apoplexy, like cerebral, was, till recently, regarded as the direct result of increased contractile force in the corresponding ventricle. This accident, for such it is, most frequently occurs in connexion with mitral obstruction or inadequacy; but, inasmuch as either of these conditions of the mitral opening is prone to react upon the right ventricle by causing hypertrophy of its walls, the latter change, having usually been accomplished before the occurrence of pulmonary apoplexy, came to be regarded as its sole cause.

This theory, however, I believe to be ill founded. In four only of the thirteen cases of mitral narrowing in which I examined the body after death, was pulmonary apoplexy found to be present (cases of Anne K., Jane M., Jane Q., and Thomas W.). In two of these the hypertrophy of the right ventricle was very slight; in the third no note was made of the state of that chamber; whilst in the fourth the right ventricle was dilated and actually thinned. Conversely, in two cases (cases of R. M'B. and T. N.), right ventricular hypertrophy existed in a high degree; and in one of these (the former) it was extreme; yet pulmonary apoplexy had not occurred.

From the data supplied by my own statistics, I therefore feel

warranted in concluding that no necessary connexion exists between pulmonary apoplexy and hypertrophy of the right ventricle. But whilst, in four only out of thirteen cases of mitral narrowing, or a fraction less than one-third of the whole number, pulmonary apoplexy was found to have occurred, hæmoptysis had actually taken place in eleven, and, to all appearance, irrespectively of the condition of the right ventricle.

In relation to another subject, which has been recently engaging the attention of experimental pathologists, namely, the alleged connexion between pulmonary infarctus and extravasation, on the one hand, and thrombosis of the pulmonary artery and right ventricle upon the other, my statistics afford some useful information.

It would seem from the experiments and observations of Lefeuvre,* that obstruction of the artery of supply to a district of tissue, will cause immediate engorgement of the capillaries and general turgescence of the part. According to Feltz† this is due to reflux from the veins and paralysis of the capillaries, and is frequently attended with extravasation of blood. In pulmonary apoplexy, therefore, and in hæmoptysis of cardiac origin, thrombosis of the pulmonary artery or its branches, or of the right ventricle, might reasonably be looked for. In all four cases of mitral narrowing in which pulmonary apoplexy was found, thrombosis of the right ventricle and trunk of the pulmonary artery existed. In two cases of pulmonary apoplexy in connexion with mitral regurgitation, in which the valves were found inadequate, without stenosis of the orifice, right ventricular and arterial thrombosis likewise existed. On the other hand, in several examples of thrombosis of the right ventricle and pulmonary artery, which have come under my notice, pulmonary apoplexy did not exist. It would seem, therefore, that whilst pulmonary apoplexy, whether associated with obstruction or valvular inadequacy at the mitral orifice, is necessarily connected with, if not dependent upon, thrombosis of the right ventricle and pulmonary artery, the latter condition, in the absence of mitral lesion, may exist without pulmonary apoplexy,

* *British and Foreign Medico-Chirurgical Review*, October, 1871.
† *Loco citat.*

or, in other words, it does not of necessity lead to this complication.

The connexion between hæmoptysis and right cardiac thrombosis would appear to be less intimate. Thus, hæmoptysis had occurred in eleven out of thirteen fatal cases of contracted mitral opening, whilst of these, thrombosis of the right chambers was found only in nine; and in only five out of eight cases of simple mitral inadequacy hæmoptysis had occurred, whilst right ventricular thrombosis existed in seven.

Hæmoptysis was, therefore, in numerical excess of thrombosis in mitral narrowing; whilst the reverse was the case in mitral inadequacy.

From the preceding abstract, the inference may be legitimately drawn, that the connexion of pulmonary apoplexy, and of hæmoptysis, with thrombosis of the right side of the heart, is not the same; pulmonary apoplexy seeming to require it as a necessary antecedent condition; whilst hæmoptysis, though generally associated with thrombosis in the last moments of life, as a further consequence of capillary infarction, may, and frequently does occur independently of it at an earlier period of the fatal illness, and as a direct consequence of the primary cardiac lesion. It may be further inferred, that as thrombosis of the right side of the heart and pulmonary artery is a necessarily and proximately fatal occurrence, apoplexy of the lung, as a consequence of it, is an event of the last few hours of life.

Fatty degeneration is a change consecutive to hypertrophy, rather than a consequence of it.

The relative inadequacy of cardiac nutrition, by which hypertrophy of the heart, when it exceeds a certain limit, is necessarily followed, added to the absolute impairment of nutrition which results from dilatation of the aorta, and atheromatous transformation and rigidity of the coronary arteries, at a later period leads to fatty degeneration and failure of the heart. The advent of this condition, which will be discussed in detail further on, is announced by weakness and irregularity of pulse, occasional vertigo, or even partial syncope; on suddenly assuming the erect posture, or making ordinary exertion; frequently, but not always, by tortuosity and rigidity of the superficial

arteries; and by weak impulse and irregular action of the heart as to rhythm and force; the sounds at the apex being weak, and when not associated with murmur, either muffled or sharp, according to the thickness of the walls of the left ventricle. At the base the first sound is usually of the same character as at the apex, but it may be clearer; whilst the second sound, owing to dilatation of the aorta, or atheromatous change of its walls, is invariably sharp and clear when not masked by murmur.

The ultimate consequences of dilatation and debility of the ventricles soon make their appearance; viz., engorgement of the lungs, and of the right side of the heart and liver, congestive bronchitis, general venous distention, anasarca, diminished secretion of urine with albuminuria, and, finally, effusion into the serous cavities.

The muscular structure of the heart may be found to present the condition of "granular" degeneration, fatty transformation, or "obsolescence" of the fibres; and frequently all three conditions or degrees of tissue-degradation are met with in the same organ or chamber.

Gangrene of distant portions of the body is never a result of hypertrophy of the heart, as such; but it not unfrequently arises from *accidents* of hypertrophy, namely, thrombosis, or embolism. Inasmuch as these formidable complications of cardiac and vascular disease will form the subject of a special chapter, it is unnecessary to allude to them further in this place.

Is hypertrophy *curable ?* I have already stated that in the absence of its complications, causal and consequential, hypertrophy of the heart should not, in my judgment, be regarded as a disease; and that, even when associated with disease by dependence or by cause, it constitutes, in the proper sense, no essential portion of the morbid condition, but is rather the product of an effort of compensation, a *nisus naturæ*, to supplement inadequacy of propulsive power in the heart. It constitutes, however, a border condition of proclivity to disease, leading necessarily, in time, to degeneration of the heart, and inviting acute inflammation of other organs, especially the lungs; which, when established, it most seriously aggravates. Hypertrophy of the heart, being in most cases secondary to organic and incurable

disease, is itself irremovable, save in the comparatively few examples where it is primary; and even here it is only partially remediable, because the calibre and thickness of the arteries have become adapted by usage to the exaggerated propulsion of the ventricles, and are not alterable by treatment.

Hypertrophy is, therefore, not strictly curable in any of its forms; and even were it removable by treatment, to effect its cure would be manifestly undesirable, in view of what has been just stated. Yet Laennec maintained that hypertrophy, whether simple or dilated, was the most curable of all the organic diseases of the heart.[*]

Hope was of opinion that in "nearly" all cases where the patient is under forty years old, of good constitution, and free from valvular disease, obstruction of the aorta, adhesion of the pericardium, and softening of the heart, hypertrophy "may be radically cured."[†] No doubt, under the conditions mentioned, hypertrophy would be found most amenable to treatment directed to its alleviation, and to the arrest of its progress; but even here it is not "radically" curable.

Hypertrophy in the primary form, or that which results from over active and long continued physical exertion, or from habitual emotional excitement, is alone even partially remediable. Nevertheless, the accidents and the consequences of hypertrophy, by whatever cause produced, are only in a limited degree under the control of medicine.

Corvisart approved of the "depletory" system of Valsalva and Albertini, which consisted in repeated bleedings and low diet, as applied to "active aneurism;" but for other forms of the affection he considered it unsuitable.[‡] He regarded Morgagni's plan of diverting blood from the heart by plunging the extremities repeatedly into hot water, although useful in alleviating the paroxysms of dyspnœa occasionally witnessed in the progress of hypertrophy, as of only temporary effect in the treatment of the affection itself. Corvisart, nevertheless, advises moderate abstraction of blood in the treatment of all forms of hypertrophy. I

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[*] *Auscultation Médiate,* tom. ii., p. 732.
[†] *Opus citat.,* p 289.
[‡] *Opus citat.,* p. 141.

cannot help thinking that the reduction in the rate and force of the pulse under Valsalva's treatment, was misinterpreted by the physicians of that time, unacquainted as they were with auscultation and percussion, as evidence of decrease in the volume of the heart.

This remark will certainly not apply to the illustrious Laennec, nor indeed to his scarcely less distinguished compatriots and cotemporaries, Bertin and Bouillaud; all of whom give an unqualified sanction to Valsalva's treatment, but with grave misgivings that patients will be found to possess the courage and perseverance to submit to it for months, and even, with brief interruptions, for years, as Laennec suggests may be necessary.

Laennec ventured to promise a large measure of success from this plan of treatment, if it be applied early, and before the grave general effects of hypertrophy have supervened. He commenced with a general bleeding to the verge of syncope, and repeated this every two, four, or eight hours; or less frequently, according to the effect, till palpitation ceased, and the impulse was reduced in force. The food should be, at the same time, reduced to one half the usual allowance; and even still further if the patient's strength exceed what is barely necessary for a few minutes' walk. For a vigorous adult, his ordinary allowance was fourteen ounces of solid food per day, two ounces of which consisted of white meats; and if soup or milk was taken, four ounces of either counted as one of solids. No wine was allowed. If, at the end of two months, palpitation and strong impulse no longer existed, " the intervals between the bleedings might be prolonged, and the severity of the regimen somewhat relaxed "* Not even the existence of dropsical effusion would deter him from the practice of this heroic treatment. In addition, he prescribed diuretics and drastic purgatives.

Bouillaud, in the treatment of moderate hypertrophy in a man of medium strength and appetite, would practice three or four bleedings from the arm, each amounting to three or four *palettes*, to which he would add in the course of treatment, one or two cuppings, amounting to two or three *palettes*. He would also prescribe digitalis, which he designates the " veri-

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* *Traité de l'Auscultation Médiate*, tom. ii., p. 783-4.

table opium of the heart." This latter medicine he uses by dusting the blistered surface of the precordium with six to fifteen grains of the powder daily; being of opinion that it acts as a local sedative.*

The practice of these two eminent men, thus given in summary, will suffice to illustrate the authorized treatment of hypertrophy of the heart at the period when they wrote. Yet who would be bold enough to pursue it, even in a mitigated form, now?

Hope's plan of treatment might be represented as the preceding, but in a less heroic form. He recommended that two to eight ounces of blood should be drawn every two to six weeks, according to the age and strength of the patient. If there had been threatening of apoplexy, the preceding quantity of blood should be taken, by means of cupping glasses, from the nape of the neck. The diet should be regulated and restricted, especially for the plethoric; and it should consist, for the first few months, of white fish and farinaceous and vegetable food. Drink should be restricted, and alcoholic stimulants strictly prohibited. Diuretics and hydragogue purgatives should be administered, especially in presence of dropsical effusion. If the patient were anæmic or feeble, iron and tonics might be necessary; and in case nervous palpitation existed, sedatives, such as opium, hyoscyamus, and digitalis, would be demanded. Under this plan of treatment, continued from one to two years, he declared that his recoveries were numerous.†

Walshe limits blood-letting in hypertrophy to the application of four or five leeches over the heart, having very properly the fear of anæmia before his mind. He especially recommends aconite as a tranquillizing agent.‡

I believe Dr Latham was the first writer who pointed out the evil effect of the active depletory treatment of hypertrophy; especially of general and repeated blood-letting. He showed that the state of anæmia thereby induced, by rendering the heart morbidly susceptible of ordinary impressions, more than neutralized

* _Traité Clinique des Maladies du Cœur_, 1835, tom. ii., p. 460.
† _A Treatise on the Diseases of the Heart and Great Vessels_, 1839, p. 284-9.
‡ _Ibid._, fourth edition, 1873, p. 297-8.

the tranquillizing action of the measures employed; and by inducing debility, favoured the occurrence of dilatation of the heart.[*]

In discussing the treatment of the consecutive hypertrophy of aortic valve inadequacy, Sir Dominic Corrigan[†] incidentally and ably exposes the dangers resulting from the active depletory system of treatment of all forms of hypertrophy.

Doctor Stokes no less strongly deprecates this plan of treatment, as likely to lead to the twofold evil result of softening and dilatation.[‡]

The two last named physicians recommend the occasional application of a few leeches to the precordium, and the avoidance of all causes of excitement of the heart. If the patient complain of constriction of the chest, with oppression and dyspnœa, and exhibit at the same time a full and bounding pulse, Sir D. Corrigan advises a moderate venesection. Dr. Stokes recommends the use of mercury where the liver is engorged in a gouty subject. He likewise advocates the use of digitalis, in doses of $\mathfrak{m}$ x–xv of the tincture thrice daily, as a tranquillizing agent in the nervous palpitation of hypertrophy.

Recent experimental inquiry into the action of digitalis upon the heart, and, indeed, my own experience of the drug in the treatment of cardiac disease, would not sanction this use of it. It is essentially a cardiac tonic and stimulant, imparting power to the heart, and regularity to its action. In the treatment of hypertrophy, therefore, it is admissible only where weakness and irregularity exist; namely, in the condition of softening and dilatation.

The treatment of *primary* hypertrophy, whether due to physical or moral agency, should consist, in the first instance, in the adoption of measures against continued or repeated excitement of the heart. Long or rapid walks, or over active exercise of any kind, should be accordingly avoided; as likewise the use of alcoholic stimulants, and mental or emotional excitement. Nevertheless, open air exercise, even of an active kind, but within the limits of moderately quickened circulation, should be taken,

* *Lectures*, No. xxxi
† *Edinburgh Medical and Surgical Journal*, vol. xxxvii., 1832.
‡ *The Diseases of the Heart and the Aorta*, 1854, p. 346.

in order to prevent derangement of primary digestion, and to promote renovation of tissue. Whilst active measures, designed to reduce the volume of an already enlarged heart, should be studiously avoided, as calculated to lead to degeneration of its structure, paroxysmal excitement or palpitation should be controlled by the application of a few leeches to the precordium, if a feeling of oppression, and some difficulty of breathing, with or without headache, and a full and strong pulse, be associated with the palpitation. But if the cardiac excitement be chiefly nervous, as indicated by a pallid and anxious face, with dilated pupils, quick, but not strong pulse, and a thrilling vibration communicated to the hand placed over the heart, then the proper remedies would be morphia with hydrocyanic acid : ℳ x–xv of the solution of the former, acetate or hydrochlorate, with ℳ ij–v of the latter, of Pharmacopœial strength, in half a wineglassful of water, given every hour until excitement be subdued and relief obtained. Veratrum viride may be likewise prescribed with advantage in similar cases, from ℳ v–x of the tincture in spearmint water every second hour, till the desired effect is produced, its action being carefully watched.* Better than either of these is the etherial tincture of Indian hemp, in doses of ℳ x–xxx suspended in water by means of mucilage, and given every second hour, if necessary, under careful supervision. Aconite may be likewise given in similar cases with good effect ; it should be administered in doses of ℳ v of the Pharmacopœial tincture every third hour, until the rate of pulsation subside. In sthenic palpitation, the bromides of ammonium and potassium may be given singly or combined, in gr. x doses, at intervals of a few hours, with the best results ; but if cardiac debility exist, I would not prescribe these salts. Digitalis would be the remedy in case of palpitation, characterized by feeble impulse, with irregular action of the heart. It should be given in doses of ℳ x–xx of the tincture, combined with ℳ v of spirit of chloroform every third hour, during the continuance of cardiac excitement. The administration of a full enema, with oil and spirits

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* Bullock has recently obtained from this agent two alkaloids, *viridia* and *veratroidia*, which are eminently depressant of the heart's action, both in regard to force and rate, and may be administered subcutaneously.

of turpentine, often materially aids in relieving palpitation
when the bowels are confined and inflated. The diet should be
nutritious and non-stimulating, containing a large percentage
of nitrogenous aliment, and a diminished proportion of fat and
hydro-carbons generally; the object to be held in view in pre-
scribing regimen and diet being, not a reduction in the volume
of the heart, but the maintenance of it in a state of structural
integrity, and the avoidance of fatty change. I have not tried
acetate of lead in the treatment of hypertrophy, with the object
of reducing the volume of the heart; nor, indeed, would I expect
satisfactory results from its use.

The treatment of hypertrophy in the consecutive form will
depend upon the particular cause to which it owes its origin.
Thus, hypertrophy of renal origin requires for its treatment
measures directed to the disease of the kidneys almost exclu-
sively. When, however, in the latter stages of the affection, the
left ventricle, by deterioration of its structure, begins to yield
under pressure from within, symptoms indicative of over-re-
pletion of the left side of the heart, passive congestion of the
lungs, and engorgement of the right chambers are exhibited.
A feeling of pectoral constriction with dyspnœa is now experi-
enced; the breathing is quickened and less deep; the carotids
throb forcibly; the great veins of the neck are engorged, and the
pulse is quick, slender, and irregular. Under these circumstances,
iron and digitalis, or the triple compound of iron, strychnia, and
quinine, is especially indicated. If the evidence of dilatation
and distention of the chambers be in excess of that of muscular
and neurotic failure of the heart, digitalis is the appropriate
remedy. I usually prescribe ♏x–xv of the tincture, with an
equal quantity of the tincture of perchloride of iron, and of
chloric ether, or spirit of nitrous ether, every three or four hours.
The latter is generally preferred for its diuretic action. Where,
however, the pulse is feeble and irregular, without characteristic
respiratory distress or venous congestion, the heart's action being
rapid and weak, with unequal and abortive contractions of the
left ventricle, not represented in the radial pulse, occurring from
time to time, strychnia and quinine should be preferred, and
are best given in combination with iron in small doses. Thus:

℞. Liquor strychniæ, Acid. sulphur. dil., aa. ℳ xxx ; Syrupi limon , ʒiv ; Sulphatis quiniæ, gr. xvj ; Sulphatis ferri granulat., gr. iv ; Aquæ font., q.s. ad ℥viij. Half an ounce to be taken every third hour.

Half an ounce of whiskey or brandy diluted with water, or half a pint of good bitter beer, should be likewise allowed twice or thrice daily , because, in addition to their stimulant effect upon the heart, they possess decidedly diuretic properties. Where engorgement of the superficial veins exists, there is generally, likewise, congestion of the kidneys and liver. Hence cupping of the loins, with or without abstraction of blood, and followed by the application of a linseed poultice, would be advisable as a means of promoting the secretion of urine, and thereby reducing cardiac and vascular pressure. With a similar object in view, drink of any kind should be allowed only in very small quantity, but often if necessary ; and, as a means of unloading the vessels generally, and those of the liver in particular, saline aperients and resin of podophylline, the latter in gr. ¼ to ⅓ doses, with compound colocynth or aloetic pill, should be administered from time to time.

The treatment of hypertrophy dependent upon valvular disease, resolves itself into that of the accidents of the valvular lesion present, and of the attendant dilatation. From the strain occasionally put upon it by injudicious exercise or muscular effort, a heart the subject of valvular disease and hypertrophy, with dilatation or fatty metamorphosis, may act inordinately.

The palpitation thus produced is best treated by the application of two or three leeches over the base of the heart, followed by a warm cataplasm, a moderate opiate being given immediately afterwards. An opiate plaster should be subsequently applied over the heart as a mechanical support and sedative. The intercurrent symptoms due to dilatation have been already discussed ; the treatment then sketched, which it is unnecessary to recapitulate here, is equally applicable in the circumstances now under consideration.

The following cases will serve to illustrate the various forms of hypertrophy described in the preceding pages, and the treatment applicable to each form of the affection.

Case XXI.—*Albuminuria; General Dropsy; Orthopnœa; strong Impulse of Heart; Hœmatemesis; Sudden Death. Cirrhosis of the Liver; Fatty Degeneration of the Kidneys; "Concentric" Hypertrophy and Granular Degeneration of the Heart.*

John O'D., aged forty-four years, a discharged soldier of intemperate habits, who had served several years in India and other warm climates, was admitted January 8th, 1866. Four months previously his attention was arrested by frequent calls to pass water. A month subsequently he noticed a swelling of the ankles, and, shortly afterwards, of the lower extremities generally, and of the abdomen. When admitted, he was generally dropsical. The lower extremities and genitals were very much distended; there was likewise ascites and flatulent distention of the bowels. Ramifying over the left side of the abdomen were several large, tortuous, and distended veins, the colour of which contrasted strongly with that of the skin, which was of waxy whiteness. The face, chest, and arms were likewise œdematous. Respiration was much embarrassed, and the pulse was quick, but regular, and rather strong. The heart pulsated with considerable force in the usual situation; its sounds and rhythm were normal; but precordial dulness was extended. There was orthopnœa. The urine was highly albuminous, sp. gr. 1010; reaction acid, and under the microscope the precipitate exhibited hyaline tube casts, some simply destitute of epithelium, and others studded with fat granules. Respiration was loud on both sides, except posteriorly and inferiorly on the right, where it was feeble and accompanied with râles. Here, likewise, was percussion-dulness; but elsewhere there was normal resonance. There was manifestly renal disease. That the liver was likewise the subject of disease, probably cirrhosis, was inferred from the great disproportion between the ascites and the dropsical effusion elsewhere; from the distended state of the superficial abdominal veins, showing visceral obstruction beneath; and from the previous intemperate habits of the patient. Finally, hypertrophy of the heart was diagnosed from the abnormal extent of precordial dulness, and the strong impulse at the precordium. Elaterium was tried; but the stomach revolted against it.

On the night of the 10th he vomited a large quantity of dark blood ; and on the following morning was found breathing stertorously, the pulse irregular and intermittent, and the impulse of the heart imperceptible. Effusion was manifestly taking place into all the serous cavities, including the pericardium. He was quite conscious. In the attempt to sit up in bed for the purpose of describing his sensations, he suddenly died in a state of syncope.

The peritoneal cavity was found distended with serum ; the liver in a most advanced stage of cirrhosis ; and the spleen enlarged, its capsule thickened and adherent to the diaphragm, and its artery tortuous, and nearly as large as the arteria innominata. The left kidney was somewhat enlarged, and, on section, presented a mottled appearance. The cortex was rather increased in thickness, and examined microscopically it showed fatty disease in an advanced stage. The pyramids were enlarged. The right kidney resembled the left in general appearance, but was much smaller, scarcely exceeding in size the suprarenal capsule of the fœtus.

The pericardium was full of serum. The heart was hypertrophied on the left side, rather pale in colour, and loaded with superficial fat. The right cavities and the tricuspid orifice were dilated, the latter admitting four fingers to pass through it with facility ; the walls of the right ventricle were much attenuated. The left auricle was rather thickened, and the left ventricle "concentrically" hypertrophied. The papillary muscles were likewise thickened. The cavity of the left ventricle was reduced in capacity. The valves were everywhere healthy.

The pleural cavities were full of serum. The right lung sank in water, but admitted of inflation ; the left lung floated.

The following measurements were taken five days subsequent to the patient's death, and after much and forcible stretching of the ventricular walls :

Heart	...	Circumference at base of ventricles, $11\frac{3}{4}$".
Aorta	...	Circumference, two inches above valves, $3\frac{1}{16}$".
Left ventricle		Wall, at apex, $\frac{3}{4}$" thick.
		„ middle, $1\frac{1}{8}$" „
		„ base, $1\frac{1}{4}$" „
		Cavity, length from aortic ring to apex, 3".
		„ width from middle of septum to opposite wall, $1\frac{1}{2}$".
		Circumference at base, $10\frac{1}{2}$".

Left auricle   ...   Wall in central portion, ¼″ thick.

Right ventricle { Wall, at apex,   ⅜″ thick.
                  ,,   middle, ¼″   ,,
                  ,,   at base, ¼″

A section of the wall of the left ventricle, examined under the microscope, exhibited a "granular" condition of the muscular fibre.

Hypertrophy of the left ventricle in this case was, in my judgment, consecutive to renal and hepatic disease, and "simple" in the first instance. Granular degeneration of the substance of the heart next took place; and subsequently the right ventricle, weakened by this change, underwent dilatation under the increased blood-pressure caused by pleural effusion and obstructed pulmonary circulation. The left ventricle, already hypertrophied, was "surprised in the full vigour of contractility" by failure of the right ventricle and stoppage of the circulation, in the effort of the patient to sit up.

The state of "concentric" hypertrophy I therefore regard as consecutive, in this instance, to the "simple" form; and as a product, not of a pathological process, but of the mode of dissolution.

CASE XXII.—*Albuminuria ; General Dropsy ; Diarrhœa ; Jaundice ; Erysipelas, and Gangrene ; Hæmoptysis ; Death. Fatty Disease of the Kidneys, Liver, and Spleen ; Congestion of the Lungs, and Pulmonary Apoplexy ; Hypertrophy and Dilatation of the Left Ventricle ; Dilatation of the Right Ventricle ; Advanced Fatty Degeneration of the Heart.*

Thomas M'K., aged forty-four years, coachman ; of temperate habits ; admitted May 5th, 1866. Two months previously, up to which period his health was excellent, he caught cold from exposure after working in the heated atmosphere of a conservatory, and was troubled with cough.

On admittance, he suffered from great dyspnœa; there was œdema of the face and feet, and the heart's action was laboured, and felt at the scrobiculus cordis. Pulmonary resonance was exaggerated; and respiration was feeble and accompanied with râles all over the chest; expectoration copious and frothy; urine, sp. gr. 1·020 and albuminous. Diagnosis: pulmonary emphysema with

bronchitis, and engorgement of the right heart and kidneys. The treatment consisted in blisters to the chest; stimulant expectorants, including tincture of larch bark and turpentine punch;* and, subsequently, quinine, and good diet. Discharged, May 23rd, so much improved as to be able to resume his work.

Readmitted on the 26th June. There was then much œdema of feet and legs, and also slight puffiness of face. Respiration was embarrassed; urine, sp. gr. 1·015, loaded with albumen, and, of late, occasionally likewise with lithates. After a few days the scrotum and penis became swollen, and required puncturing. About half an ounce of clear serum, collected from the punctures, was tested by ebullition the following day and found to contain only a trace of albumen. Astringents were employed to check diarrhœa which was occasionally present; blisters were applied to the chest; turpentine punch was administered; and, subsequently, iodide of potassium in infusion of cinchona.

July 12th. Pulse 114, and barely to be felt; respiration 66. Both sides of the chest were dull half way up, and to the same extent respiratory sounds were absent; sputa rust-coloured and viscid; lower limbs and genitals swollen. The chest was wrapped in a warm poultice, and whiskey punch was given.

July 17th. Pulse 114; respiration 42. Patient remarked that the rate of his respiration sometimes varied; ascites; genitals much swollen, and prepuce punctured. Baillie's pills were given twice daily.

July 21st. Has been taking tinct. digitalis in ℳ x doses, with spirit of juniper, for the last two days, notwithstanding which there is all but complete suppression of urine. Œdema has increased, and now extends to hands. Pulse scarcely to be felt, and not to be counted at the wrist. To have grs. xv of compound powder of jalap, with gr. $\frac{1}{12}$ of elaterium twice daily.

July 23rd. Patient is deeply jaundiced. Elaterium has acted twice, and penis is less swollen. The entire of the left lower extremity, to the hip, is in a state of erysipelatous inflammation, threatening gangrene. Lead lotion was directed to be kept constantly applied to the inflamed surface.

* Known in Dublin as "Parr's Punch," and made with whiskey and spirits of turpentine, in equal proportions, diluted with sweetened hot water to the proper strength.

July 25th. The entire of left lower extremity is in a state of gangrene. From the knee downwards the limb is studded with large bullæ, and the cuticle detached. The thigh is red and tense ; and inflammation has extended to the scrotum, which, on each side, is passing into a state of gangrene. Patient all but pulseless ; but is quite conscious and free from suffering ; face deeply jaundiced and shrunken.

July 26th. Entire left leg and thigh dark and sloughy ; scrotum likewise in a state of gangrene, which extends in dark streaks over the front and upper portion of the right thigh. Respiration tranquil, and patient still hopeful.

28th. Spat up a good deal of blood last night, and died at half past nine o'clock this morning.

Post mortem examination seven hours afterwards. There was copious effusion into all the serous cavities. Kidneys large, fatty, and smooth on the surface ; the cortex was much thickened, and of a light yellow colour ; minute oil granules, as exhibited under the microscope, were interposed between the tubes and blood vessels ; the epithelium and gland structure, however, seemed healthy. The liver was large, yellow, and readily torn ; and, examined microscopically, the hepatic cells in most instances were found to contain eight to ten highly refracting oil spherules ; large oil drops were likewise dispersed over the field of vision. Spleen fatty, and fissured.

Lungs healthy structurally ; but bases were congested and solid, and smooth dark and dry in section ; they floated in water. In the anterior edge of the right lung was a nodule of blood as large as a bantam's egg ; in a corresponding situation in the upper lobe of the left lung, and also in the upper portion of its inferior lobe, were similar but larger collections of extravasated blood.

The heart was large, and, with one inch of the aorta and pulmonary artery attached, weighed fourteen and a-half ounces. The right cavities contained some dark blood ; their walls were soft and thinned. Left cavities much dilated, and walls slightly thickened. Valves all absolutely healthy. Aorta of ordinary size, and its coats healthy. Heart flabby, and, when laid on the table, was flattened out by its own weight. On section the walls

presented a yellow hue, and a portion examined microscopically presented a good example of fatty degeneration of the muscular substance, the fibres consisting of linear rows of minute circular refractile dots about 10,000th part of an inch in diameter. These were likewise abundantly scattered in a free state over the field, as if by liberation from the sarcolemma.* The outline of the fibres was quite distinct, but, with the exception of a few places where it was very faintly visible, transverse striation was entirely obliterated

From the outset, I regarded this case as an example of primary disease of the kidneys, with consequent hypertrophy and consecutive fatty disease of the heart. It is probable, however, that fatty change of the heart and kidneys was cotemporaneous: the former supervening upon a state of simple hypertrophy, and the latter upon one of sub-inflammatory or congestive enlargement. The liver and spleen would seem to have undergone the fatty transformation at a later period, under the influence of the general impairment or perversion of nutrition. Pulmonary apoplexy and hæmoptysis were probably the result of mitral regurgitation by yielding of the walls of the left ventricle, dilatation of its cavity, and consequent reflux by inadequacy of healthy valves, shortly before death. The jaundicing is of great diagnostic value in such cases, as serving to distinguish structural disease of the liver from simple congestion.

CASE XXIII.—*Oppression and Palpitation supervening on emotional shock; Paroxysmal Dyspnœa; Hypertrophy with Dilatation; Death. Fatty Degeneration of the Heart; Dilatation of the Aorta.*

Peter S., a gentleman farmer from the county of Meath, aged fifty-six years, was admitted into hospital as a private patient, May 30th, 1866. Seventeen months previously, after some severe family afflictions, he experienced rather suddenly, and for the first time, oppression of the chest and palpitation, accompanied by nervousness and great anxiety. Since that date he has been subject to occasional attacks of dyspnœa with restlessness and anxiety. Has likewise had from time to time a feel-

* I am convinced that the muscular fibres of the heart possess a sarcolemma.

ing of great debility and sinking, not amounting, however, to syncope.

Is rather fresh in colour; pupils dilated, and face expressive of fear and anxiety; slight œdema of feet and legs; pulse 104, and remarkably weak but regular. Chest universally resonant. On right side respiration is feeble, and accompanied in supra-mammary region with slight crepitant râles; on left side it is puerile.

Precordial dulness extended; impulse of heart strong at apex, and here the first sound is obscure. Over the base and at mid-sternum, there is a murmur with, and masking, the second sound; but it is not transmitted more than an inch above the aortic orifice. The carotids pulsate visibly, but not the temporal or radial arteries. Fauces congested. No appetite for food. Orthopnœa. Sleep disturbed by unpleasant dreams and startings.

Diagnosis: dilatation of the aorta, hypertrophy, and probably fatty degeneration of the heart. To have Hoffman's anodyne and Battley's sedative in moderate doses with camphor water every third hour, and a little brandy and water occasionally. On the 1st of June, after a rather tranquil night, and whilst the patient was under examination, the breathing was noticed to be singularly irregular; it was quick, loud, and suspirious; but immediately the examination of his chest was suspended, it became slow, and was entirely suspended during a period of several seconds. Tongue loaded; stomach and intestines inflated. To have pil. rhei co., gr. x. Stop brandy. He died on June 11th, manifestly from inanition and exhaustion.

Post mortem twenty-four hours after death. Much liquid in pericardium. Heart flabby and enlarged, weighing twenty-seven ounces, with an inch of the aorta and pulmonary artery attached. There was a good deal of fat on the surface of the heart; and at the apex, and partially embedded in the muscular substance, there was a solid mass of fat one-eighth of an inch thick.

The anterior surface of the right ventricle presented a large "milk spot." The right chambers were distended with dark co-agulated blood; they were greatly dilated, as was likewise the tricuspid orifice. The walls of the right ventricle were somewhat

thinned. The left ventricle was much thickened and dilated. Aorta greatly dilated for some distance above orifice, and its walls slightly atheromatous. Valves all structurally healthy. Microscopically examined, the muscular structure of the heart presented the following appearance. The outline of the fibres was well defined, and the transverse striæ were pretty well seen; but in several of the fibres, and in the direction of their long axes, minute transparent dots were visibly disposed in linear series. In these situations the striæ had disappeared.

There had been total loss of appetite for several days preceding death, the patient absolutely refusing solids of any kind, and taking only an infinitesimal quantity of liquid nutriment in the form of beef-tea with a little brandy. It seemed as if the tissues of the body had ceased to assimilate, long anterior to somatic death. The tongue, during this period, was dry, brown, and crusted.

As to pathological sequence, I incline to the opinion that there was primary atheroma of the aorta, and consequent left ventricular hypertrophy; and that from this twofold cause arose sudden dilatation of the aorta by overstrain, on the occasion of the emotional excitement already mentioned.

The diagnosis of dilated aorta rested mainly upon the phenomenon of paroxysmal dyspnœa, which I regard as pathognomonic of this special condition. But on this subject generally I will state my views more in detail in a future chapter.

CASE XXIV.—*Rheumatism; Systolic Apex-Murmur; Death. Hypertrophy of Left Ventricle, and incompetence of Mitral Valve from Rupture of Tendinous Chords; Pulmonary Apoplexy.*

George P., aged thirteen years, admitted November 7th, 1866, under Dr. Hughes, and came under my notice on the 15th whilst I was doing temporary duty for him. The boy had rheumatic fever three months previously, from which he made a satisfactory recovery; but, shortly afterwards caught cold by going out without his boots on a wet day, and has been suffering from cough and violent palpitation since that time.

When first seen by me, his face was bloated and slightly con-

gested, the cervical veins distended, and the external jugulars pulsating synchronously with the heart; supra-clavicular fossæ obliterated; chest barrel-shaped and nearly motionless. Percussion-resonance was exaggerated, and respiration loud over the front of the chest. Cardiac impulse strong, and precordial dulness extended to right side. Fremitus over extreme left apex; second sound sharp and clear, but first sound replaced by a loud bellows-murmur, terminating with a faint, squeaking, musical note, resembling that elicited by blowing through a reed. At the base of the heart, and over the front of the chest generally, both sounds were heard; the second normal, and the first represented by a bellows-murmur similar to, but less loud than, that heard at the apex, and louder in the course of the pulmonary artery than in that of the aorta. The base of the right lung was dull posteriorly, and over the entire posterior surface of the chest a loud systolic murmur was audible. Liver smooth on surface, and extending below level of umbilicus. Respiration quick, shallow, and almost exclusively abdominal; and respiratory movement confined to left side, where it was very active. At base of thorax there was a circular constriction, corresponding to the attachments of the diaphragm and apparently caused by its action. There was dry cough, with occasional expectoration of blood-stained mucus. Pulse 120, weak, and dicrotous. No visible pulsation of arteries. Urine, sp. gr. 1·030, slightly acid, and free from albumen; feet œdematous.

I ceased to have charge of the patient on the 20th November. The entire body became subsequently anasarcous in an extreme degree; and for the last few days of life, the face and neck turgid, and the extremities livid.

Death took place on the 14th of December.

Post mortem six hours after death. Both pleural cavities were full of serum; the lungs were engorged but crepitant generally, and in the middle and inferior lobes of the right were two masses of extravasated blood, each the size of an orange. The base of the left lung was solid; it sank in water, and on section was dry and spleen-like, yielding neither blood nor serum. The pericardium contained some ounces of serum. The heart was considerably in excess of the normal size, smooth on the sur-

face, and free from adhesion. The right chambers contained a little dark and partially coagulated blood; the right ventricle was somewhat dilated and thickened, the tricuspid orifice and valves normal, as were likewise those of the pulmonary artery. Fossa ovalis remarkably deep and recessed, and the operculum adherent to the left surface of septum, at some distance from the margin of fossa, which was, however, imperforate, not admitting of direct communication between the auricles. Left auricle normal; left ventricle dilated and thickened, but not in a high degree. Mitral valves structurally healthy; but the free margins of both segments, to the extent of a quarter of an inch at their anterior and left extremities, had been detached from the tendinous chords, the white mamilloid fragments of which, about two lines in length, remained in connexion with the margins of the valves and the adjacent fleshy columns respectively.

Water poured into the left ventricle flowed freely into the left auricle through an aperture, as large as a pea, bounded by the pendulous edges of the valves.

The aorta was reduced in size, barely admitting the index finger from the ventricle. The aortic valves were competent, and both they and the coats of the aorta were perfectly healthy.

There was a good deal of serum in the peritoneum. The liver was large and mottled white, with intervening dark red areolæ; and on section it presented light areolæ, each surrounded by a dark red vascular surface, as in the early stage of portal venous congestion. Kidneys and spleen healthy.

The history of this boy's illness did not date back beyond three months, when he had rheumatism; this, however, left none of the ordinary traces upon the heart. Rupture of the left *chordæ tendineæ* would seem to have occurred subsequently to this attack, when the boy caught cold; as shown by the occurrence of dyspnœa and palpitation then for the first time, yet without the history of strain or other injury to the chest. In this view the case is further remarkable, as exemplifying functional narrowing of the aorta from reduction in the volume of blood circulating through it within the short period of three months.

Strong impulse of the heart, with weak pulse, is diagnostic of stenosis of the aorta. If this arise from lesion at the aortic

orifice, whether valvular or annular, there will be either a single aortic murmur, systolic in rhythm, or a double murmur, both systolic and diastolic, of similar origin. The absence of any such murmur, when strong cardiac impulse and weak pulsation at *both** wrists are detected, would warrant the positive diagnosis of functional narrowing of the aorta, such as that presented in the foregoing case. Aortic stenosis of this kind, when not the result of congenital imperfection of the interventricular septum, or of patency of the foramen ovale, is consecutive to mitral stenosis or reflux, and is therefore usually associated with either presystolic or systolic murmur at the left apex.

CASE XXV.—*Double Aortic Murmur ; Dysphagia, with Hæmoptysis; Death. Hypertrophy and Dilatation of both Ventricles; Detachment of segment of Aortic Valve ; Double Congestive Pneumonia ; Enlargement of Bronchial and Mediastinal Glands.*

John R., aged thirty years, painter's assistant, admitted November 16th, 1866 ; served as a soldier for some years, and whilst engaged in Africa in defending a stronghold against a raid of the natives, was thrown from a rampart and sustained a shock on the front of the chest. He felt stunned ; but subsequently recovered, and as he believed, perfectly. This accident occurred some years ago. He had been intemperate for twelve months preceding his admittance into hospital ; but had never been seriously ill till six months prior to that date, when he began to get thin, and had nausea and partial loss of appetite.

On the 12th of November, four days before his admission, he experienced what he described as a choking sensation in the neck, and some difficulty of swallowing, accompanied by nausea and vomiting. These symptoms lasted three days, and then all passed away except irritability of stomach, which continued some time longer.

When the man was first seen by me his legs were slightly œdematous ; the veins of the neck were remarkably congested,

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* I have in view here the possibility of embolic infarction of the brachial or radial artery on one side ; occlusion of the subclavian by the pressure of an aneurism or other tumor ; or congenital malposition or dwarfing of the radial.

and especially so on the left side. There was a loud hacking dry cough. Pulse 108, weak, but regular, and equal on both sides. Pulmonary resonance and respiratory sounds were normal over the entire front of the chest; precordial dulness was likewise normal in extent and degree. Posteriorly there was some slight dulness over the bases of both lungs; but otherwise the condition of these organs was normal. A double murmur existed at the base of the heart, systolic and diastolic in time. Of these, the diastolic murmur was relatively louder; it was transmitted through the ascending aorta, but with diminished intensity, and (for a diastolic murmur a very unusual circumstance) likewise into the great arteries of the neck, and diffused over the front of the chest generally. Posteriorly a single murmur was faintly heard at the inferior angle of the left scapula.

On the following day, 17th, I found him perspiring profusely, and complaining of pains in the ankles and knees, which were swollen. Pulse 120.

On the 18th he spat a little blood, the right side of the chest being dull posteriorly; and on the evening of that day he vomited a quantity of bile-stained liquid mixed with blood.

On the 19th the pulse had come down to 102. He was perspiring freely, and complained of pains in the ankles and calves of the legs; a single systolic murmur was audible at the apex of the heart; dulness was increased both in extent and degree over both sides of the chest posteriorly; and, over the bases of both lungs a loud muco-crepitus was heard. The temporal and cervical veins were distended. There was likewise, at this date, visible pulsation or throbbing of the anterior tibial arteries and all their branches even to the toes, accompanied with vibration sensible to the hand.

From the 19th to the 23rd, when he died, no general examination of the chest was made, owing to his extreme restlessness. During that interval he complained of undefined pains in the knees and ankles, was restless, slept but little, and had a loud spasmodic cough with continual expectoration of blood. On the day preceding his death, which took place suddenly on the night of the 23rd, it was remarked that the temporal arteries pulsated strongly.

Post mortem. Both pleural cavities were full of straw-coloured serum, and the pericardium contained a pint of similar fluid. The lungs were engorged with blood, but resonant, and floated in water, with the exception of the inferior lobe of the left. The heart was somewhat larger than in health, and globoid in figure. The right cavities were distended with dark and partially coagulated blood, the clot extending from the right ventricle into the pulmonary artery. The right ventricle was slightly dilated, and the tricuspid orifice readily admitted the points of the four fingers and thumb. The left chambers were likewise distended with dark coagulated blood, and the left ventricle was dilated, and its walls were thickened. The mitral orifice and valves were normal; the aortic orifice, viewed from the ventricle, was likewise apparently healthy, but when examined on the arterial aspect it was found that the right anterior segment of the sigmoid valve had been partially detached from the root of the aorta; its left angle, to the extent of nearly half an inch, hanging loosely into the ventricle in the natural position of the heart. Water poured into the aorta at once flowed into the ventricle through the patulous orifice. The valves were otherwise perfectly sound, and entirely free from disease, as was likewise the aorta. The bronchial glands were greatly enlarged and pressed upon the bronchi, and several large glands were found in the posterior mediastinum detruding the œsophagus to the left, and pressing upon the posterior wall of the trachea. These glands were solid, dark in colour, and mottled with white spots on the superficial surface, and likewise on the surface of section. In the centre of one of these glands there was a mass of hard cheese-like matter, which, on pressure, yielded a quantity of pus. On examination, microscopically, the cheesy substance was found to be tubercular.

There can be no doubt, judging from the man's previous history, and from the evidence afforded by the *post mortem* examination, that the rupture of the aortic valve occurred at the time when he was thrown from the rampart. It may be easily supposed that if, at such a moment, the chest was in the acme of expiration, the sternum being thereby brought into close contact with the pericardium, and through it with the root of the aorta, and if, during the diastole of the ventricle and at the moment of

reaction of the aorta upon its contents, the semilunar valves being closed, a severe shock were received upon the sternum, the valve segments might be thereby ruptured or rent from their attachments.

An accident attended with such consequences in a heart previously sound is, however, exceedingly rare ; Dr. Peacock having been able to find only seventeen examples recorded   The case is interesting, moreover, as exemplifying the existence of the physical signs of disease of the aortic valve, both obstructive and regurgitant, and dependent entirely upon an accidental lesion in the absence of disease of the heart or valves; and as showing that left scapular murmur, though usually dependent upon obstruction or regurgitation at the mitral orifice, may be caused by lesion of the aortic valve exclusively.

Temporary dysphagia with hæmoptysis may be due to causes other than thoracic aneurism, as this case proves ; and, in regard to the pulmonary complication which it presented, Dr. Stokes has well remarked upon the frequent occurrence of pneumonia of a low type in connexion with inadequacy of the aortic valve ; as well as upon the frequency with which, under such circumstances, sudden deaths occur.

As to the differential diagnosis between ruptured aortic valve and lesion of the same valve arising from disease : irrespectively of the aid afforded towards forming a correct judgment by the history of the case, the most valuable sign, and one which is in a degree pathognomonic of partial detachment of the valve, is the occurrence of *arterial thrill*, of systolic rhythm, in connexion with double murmur at the aortic orifice.   I should henceforth regard such a phenomenon, in all doubtful cases, as conclusively establishing the existence of a loose and vibrating valve-flap at the root of the aorta, where no ground for suspecting thrombosis or embolism existed.   Embolism would declare itself by other and indubitable evidence ; whilst the precipitation and attachment of a flake of fibrin at the mouth of the vessel would give rise to a thrill of only temporary duration, and would yield a murmur most probably musical in quality.   A diastolic basic murmur, audible in the arteries of the neck, does *not* belong to aortic patency from disease ; it is a phenomenon so strictly exceptional

that I have never heard it, save in this single instance. Without venturing even to suggest an explanation of the striking contrast herein presented with the murmur of inadequacy of the aortic valves from ordinary morbid change, I should in future regard it as indicative of failure of the valves from some other cause, most probably mechanical injury.

CASE XXVI.—*Irregular and Suspirious Breathing; Intermittent Pulse, and Great Debility; Polyuria; Death. Granular Degeneration of the Kidneys; Fatty Deposition on the Heart; Simple Hypertrophy of the Left Ventricle.*

John D., aged forty-eight years, labourer, admitted September 25th, 1864. For last three years he had been weak and out of health, and his feet began to swell six weeks prior to admittance. Pulse weak and intermittent, and respiration occasionally accelerated and suspirious in the recumbent posture. Urine passed in large quantity, amounting to more than two quarts in the twenty-four hours, sp. gr. 1·010, and highly albuminous; feet slightly swollen; apex-pulsation somewhat to left of normal position, and both sounds of heart sharp, clear, and free from murmur. At midsternum the first sound was distinctly double; the first element in time being dull, and the second, which immediately succeeds the former, sharp, and " clicking." These characteristics were recognizable only at the end of expiration, when the chest-wall had entirely subsided. To have citrate of iron and quinine (gr. v.) ter die.

The patient gradually became listless, and then somnolent; the stomach became irritable; the appetite ceased, and he quickly emaciated.

For the last fortnight of his life there was intercurrent diarrhœa, and during that time the breath was cold. Œdema had quite disappeared. Respiration was slow and stertorous. He died comatose, November 17th, and on the following day the body was examined. The heart weighed fourteen and a-half ounces; it presented some superficial fat at the base. Right ventricle very thin, but not dilated, and exhibiting in section a layer of fat on its external surface; some decolorized clot in right chambers. Left ventricle thickened, but not dilated; its walls measur-

ing half an inch at base and central portion. Left auricle dilated; valves all strictly normal; muscular structure healthy. Ascending aorta dilated sufficiently to admit three fingers on edge; dilatation commencing above the valves, which were competent. The lining membrane of the vessel was of a cherry-red tint not removable by washing. Kidneys reduced in size, and nodulated on the surface; the right weighed three and a-quarter, and the left three and a-half ounces. The nodules on the surface of the kidneys were large, but slightly elevated, and of much lighter colour than the intermediate surface. In section they were light yellow, as was likewise the greater portion of the cortex. The latter was increased in thickness, and passed in between the pyramids, which were dwarfed. Under the microscope, and with a power of 222 diameters, the Malpighian bodies were seen nearly filled with amorphous matter, which obscured the vascular glomeruli, and yielded negative results when treated with iodine and sulphuric acid.

The primary disease in the foregoing case was renal, which was followed by left ventricular hypertrophy, and, subsequently, by fatty deposition on the heart, and dilatation of the aorta. Simple hypertrophy of the left ventricle, the first change in the pathogenetic series consecutive to granular disease of the kidneys, would have been converted in this case, under the influence of greatly impaired nutrition, into dilatation with thinning of the walls, had the fat been deposited amongst or within the muscular fibres, in place of upon the surface of the heart; because the walls, then weakened in contractile and resisting power, would have yielded under the pressure of the blood from within. I think that such is the history of simple hypertrophy in most instances, as already stated; namely, that it constitutes the first of a series of pathological changes in the heart, the last member of which is dilatation, which accompanies or supersedes hypertrophy according to the state of general or special nutrition.

Under the same circumstances, namely, if tissue-degeneration of the heart had taken place instead of fatty deposition, the aorta would have been dilated at its orifice, and valvular inadequacy would have been the result. Dilatation of the first portion of the aorta, including the orifice or not, constitutes, in my

opinion, the essential condition for the occurrence of the peculiar rhythmical irregularity of breathing known as the "ascending and descending respiration" of Cheyne. This connexion was well exemplified in the case just narrated. Finally, the dependence of reduplication of the first sound of the heart upon *simple* hypertrophy of the left ventricle is illustrated in this case.

CASE XXVII.—*Albuminuria and Anasarca; Orthopnœa; Heaving Impulse of the Heart, and Reduplication of the First Sound, with subsequent Development of Mitral Systolic Murmur; Uræmic Convulsions and Coma; Death. Cirrhosis of the Kidneys; Simple Hypertrophy of the Left Ventricle; Recent Endocarditis of the Mitral Valve; Contraction of the Aorta.*

Marianne C., aged nineteen years, a domestic servant, admitted October 2nd, 1869. When a child she had severe pain in the region of the heart. Menstruation has been irregular during the last twelve months, and for the last two months it has been entirely suspended. Nine months ago she began to suffer from shortness of breath and palpitation, and three months since she was forced by ill health to relinquish her employment. For the last two months she has not been able to lie down, owing to the feeling of suffocation induced by the recumbent posture. A week since she spat a little blood, and about the same time her feet began to swell. When admitted she was anæmic; feet slightly puffed; there was orthopnœa, and great respiratory distress on assuming the recumbent posture even for a moment. Pain shooting down the left arm. Respiration in sitting posture 48; pulse 108, weak, but regular; occasional vertigo; urine, sp. gr 1 010, and loaded with albumen. Great increase in extent of precordial dulness; heaving impulse, and very peculiar action of the heart, consisting of a rolling motion associated with three cardiac sounds, of which the first two were systolic in rhythm, and dull. No murmur. Râles over base of right lung both anteriorly and posteriorly. To have quinine and ether, and an opiate plaster over the heart.

October 10th. Stomach has been very irritable, refusing food and medicine; dyspnœa much aggravated at night, and threaten-

ing suffocation. A distinct systolic apex-murmur now exists, and only two sounds are audible. To have tincture of digitalis with syrup of the American wild cherry (Prunus Virginiana).

25th. Since last report, feet, legs, and genitals have become greatly swollen, but are pale and wax-like. Vertigo; systolic apex-murmur of a soft and blowing character. Amaurosis and coma gradually supervened, the latter very slowly, and were succeeded by a series of epileptiform convulsions, about three in the twenty-four hours, commencing with rapid twitchings of the face, arm, and leg, first of the left side, and then of both sides, and ending with stertorous breathing and profound coma. The pupils were dilated during the stage of clonic spasm, and contracted in that of the consecutive coma. During the intervals between the fits there was partial consciousness or susceptibility to strong sensorial impressions. Deglutition was perfect; alvine and vesical evacuations involuntary.

She died on the evening of the 26th October.

The heart was enlarged, and, with an inch of aorta and pulmonary artery attached, weighed nineteen ounces. There was a large "milk-spot" on right ventricle, and one upon either auricle. The left ventricle was greatly hypertrophied, and constituted an excessive proportion of the volume of the entire organ. It was not dilated; walls about ten lines thick at base and centre, but much thinner at the apex. Mitral valve apparently competent to close the orifice, and structurally sound, save that there was slight thickening of the free edge, and crimson reddening of this, and of the entire ventricular surface of both segments.

Left auricle normal, and mitral orifice of average size. Both left chambers contained a good deal of dark clotted blood. Muscular substance of left ventricle examined microscopically, was found healthy. Aortic valves normal and competent, and aorta healthy, but in calibre somewhat under the normal standard.

Right chambers normal. Right ventricle contained a solid coagulum of a globular figure, and coated on the surface with yellow fibrin; it projected through the tricuspid orifice, and divaricated the valves, but had no connexion in the auricle. Kidneys much reduced in volume, and lobulated on the surface;

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the right weighing two and a-half, and the left three ounces. Section showed thickening of the cortex, which was dark and congested ; examined microscopically, it exhibited no abnormal appearance, except considerable thickening, with hyaline appearance of the periphery, of the Malpighian bodies. The Malpighian cones were much reduced in size.

As in the case last detailed, the starting point of disease in that just given was manifestly cirrhotic or granular degeneration of the kidneys. From this, or rather from the capillary obstruction consecutive to it, arose simple hypertrophy of the left ventricle.

The occurrence of consecutive endocarditis whilst the patient was under observation, was announced by mitral systolic murmur; and, at the examination of the body, this was proved, by the still recent inflammation of the valves, to have been coeval with the development of murmur. Disease of the kidneys was diagnosed early in the progress of the case, as was likewise simple hypertrophy of the left ventricle ; and the convulsions were regarded as uræmic.

Both this and the preceding case afford notable illustrations of the causal connexion between simple hypertrophy of the left ventricle and reduplicated first sound. In both cases this was the only cardiac lesion to account for the phenomenon.

I have stated at length in a previous chapter (p. 164) my view as to the cause of reduplication of the first sound. It consists essentially in a resolution of the sound into its two component elements of apex-shock and valve-tension. If the left ventricle be in volume greatly in excess of the remainder of the heart, without having undergone enfeeblement by proportionate dilatation, or by tissue change, it will strike the chest-wall with vigour and produce a loud but dull sound. Further, the impact of the left ventricle, owing to the energy of its contraction, would conceivably, in such a case, anticipate the tension, though not the closure, of the auriculo-ventricular valves, and so give rise to double first sound. The second element of such a sound is invariably sharp and clicking, such as membranous flaps would yield under strong and sudden tension. The disappearance of the second element of the double sound in the latter of these

cases, simultaneously with the development of mitral regurgitant murmur, tends to confirm the view just stated as to the cause and diagnostic value of reduplicated first sound.

CASE XXVIII.—*Paroxysmal Dyspnœa ; Postdiastolic Basic Murmur ; Sudden Death. Hypertrophy with Dilatation of both Ventricles ; Thrombosis on both Sides of the Heart ; Dilatation of the Aorta, and consequent inadequacy of the Aortic Valves ; Granular Degeneration of the Substance of the Heart ; Cirrhosis of the Liver.*

William H., aged forty-five years, an engineer by profession, in which capacity he served many years on board ship exposed to a temperature varying from 107° to 112° F., but latterly employed on railways, was admitted into hospital, June 26th, 1870. He stated that his habits had been temperate, and that he had been ailing two years prior to date of admittance. Within that period he often had temporary dimness of vision and threatening of syncope, and about the end of the preceding December he vomited some dark blood.

When visited by me on the morning of the 27th, he was suffering from dyspnœa of a paroxysmal character accompanied with puffing, and associated with a feeling of great oppression, referred to the region of the sternum, and expressed by frequent moaning. Feet and hands cold and livid, but not œdematous ; slight engorgement of cervical veins, and slight cough. Pulse 96, feeble but regular, and not visible. A strong and heaving diastolic impulse was perceptible throughout the precordium. Apex-pulsation in nipple line, and in sixth intercostal space, and here a (transmitted) diastolic murmur was audible, but no first sound save a weak impulse element.

At the base the first sound was of a similar character, and a loud and soft postdiastolic murmur existed ; no murmur audible in the neck. To have citrate of iron and quinine in gr. iij doses thrice daily, and chloral hydrate (gr. xv) at night.

On the 29th, his distress continuing unabated, two leeches were applied at midsternum with much relief to the feeling of oppression. At 9 o'clock P.M. he declared he felt much better,

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and at 4 o'clock the following morning he was found dead in bed.

Post mortem. Lungs congested; liver in early stage of cirrhosis, and covered with false membrane on the anterior surface to the extent of the palm of the hand. Heart enlarged and elongated, weighing twenty-three and a-half ounces. Aorta dilated, admitting readily the four fingers and thumb, and measuring one and a-half inch in diameter immediately above the valves. It presented patches of atheroma; and, with the exception of the sinuses, where the walls were apparently healthy, the lining membrane throughout was corrugated, and of a pink hue irremovable by washing. Left ventricle dilated and somewhat thickened, its walls measuring half an inch at the base, gradually thinning off towards the apex; and here, at a point about one-eighth of an inch in diameter, it was reduced to the thickness of two lines. It contained some dark coagulated blood which passed into the aorta. Mitral valve adequate and perfectly healthy. Aortic valves healthy, but incompetent owing to dilatation of the vessel. The anterior right segment was slightly thickened throughout the *lunula*, and hung on a lower level than the other two. Water poured into the aorta passed rapidly into the ventricle. Right ventricle dilated and thickened, and containing some decolorized clot, which passed from the right auricle through the tricuspid orifice, divaricating the valves, and by a narrow band, into the pulmonary artery; the valves, walls and calibre of the latter vessel were normal. Auricles normal as to capacity and thickness, the right containing much decolorized fibrin, which had completely filled the appendix, and passed into the ventricle as already described.

Tissue of heart of a less pinkish tint than natural, and in the "granular" stage of fatty transformation.

The diagnostic value of right basic postdiastolic murmur in regard to the precise state of the aortic valves, is well exemplified in the physical signs exhibited in this case, as explained by the morbid changes discovered after death. The valves were healthy with the exception of slight thickening of the free margin of one of the segments; they were, therefore, capable of yielding the tension-sound (second sound), but incompetent owing to dilatation

of the aorta ; hence the murmur succeeding the second sound. The commencement of ventricular diastole and of aortic reaction is coeval with the closure of the semilunar valves, and denoted by the second sound arising from the tension of these valves, as already explained. Therefore, if the valves be in a healthy condition, quite irrespectively of their competency to close the orifice, a normal second sound will be heard ; but if they be, at the same time, inadequate from any cause not compromising their structural integrity, or only very slightly affecting it, such as deposit upon, or thickening of, their edges, to an extent sufficient to preclude accurate adaptation of the segments ; or if they be inadequate by dilatation of the orifice of the vessel, then a murmur will follow a normal second sound. Such a murmur I designate as of postdiastolic rhythm.

To distinguish between the two causes just indicated as capable of giving origin to a murmur of this rhythm, the paroxysmal dyspnœa, more or less regularly ascending and descending in rhythm, will serve as a positive guide. As stated in my comments on a previous case, I believe this symptom to be pathognomonic of dilated aorta. When, therefore, in association with this symptom, a clear second sound immediately succeeded by a murmur is heard, the diagnosis of valves structurally healthy, but incompetent from dilatation of the aorta, may be confidently made.

The immediate cause of death in the foregoing case I believe to have been thrombosis of the right chambers of the heart, and inadequacy of the tricuspid valve by entanglement of the connecting band of fibrin. The consequence was that the left ventricle, already in a degree incompetent to maintain the arterial circulation, partly owing to its inherent weakness and partly by the excessive dilatation of the aorta, now imperfectly distended with blood from the lungs and inadequately nourished by a failing coronary circulation, ceased to contract, and death was the result of ventricular asystole.

CASE XXIX.—*Anæmia and great Debility; Urine of low sp. gr., and presenting Hyaline Casts; Dropsical Effusion; Death. Cirrhosis of Kidneys; Hypertrophy and Fatty Degeneration of Left Ventricle of Heart, and Dilatation of Right Ventricle.*

Margaret T., aged eighteen years, of respectable birth, was admitted November 29th, 1871. Had never had rheumatism or other serious illness. Her chief complaint was of debility. She was pale and undergrown, and remarkably nervous and choreic in her manner and movements. Had never menstruated.

Under a tonic plan of treatment, including iron and shower-baths, she improved so much that her discharge was contemplated, when, about three months after admittance, the feet and legs began to swell; the stomach now occasionally rejected food, and the pulse became gradually weak and filiform.

The urine was tested and found of a clear, amber colour, acid, sp. gr. 1·010, and free from albumen. The precipitate examined microscopically exhibited epithelium with a few transparent tube-casts. The face soon became swollen, and the breathing much embarrassed, whilst the pulse was hardly perceptible.

Precordial dulness seemed to be extending; no cardiac impulse could be detected, and the sounds of the heart were faint and distant, but free from murmur. The cervical arteries pulsated visibly, and the face and lower limbs became livid.

She died on the 15th March, 1872.

On examination of the body both pleuræ were found quite full of serum, whilst the pericardium contained only about one ounce. The heart was much enlarged, weighing fifteen and a-half ounces; the left ventricle dilated and thickened, but the right ventricle rather attenuated. Examined microscopically, the structure of the ventricles was found to be in the granular stage of fatty degeneration; the transverse striæ had disappeared, and the section was dotted with large oil-drops.

The valves of the heart were all normal, and the foramen ovale was closed. Kidneys lobulated on the surface and cirrhosed, but not reduced in size; the left weighing four, and the right four and a-half ounces. The cortical structure was rather thicker than normal, firm, and light in colour, but the pyramids

were reduced in size and congested. A section of the cortex examined microscopically presented the capsules of the Malpighian bodies greatly thickened, and composed of concentric laminæ of fibroid texture, whilst the glomeruli were so much dwarfed as to be scarcely discoverable; some thick puriform fluid was found in the calices. The liver was large and congested; the spleen small; no liquid in the peritoneum.

An enlarged heart, weakened by fatty degeneration, is not unlikely to be mistaken for hydro-pericardium, more especially if associated with hydro-thorax and slight effusion into the pericardium itself; all which conditions existed in this case. Trousseau adverts to the difficulty of diagnosis, even in the absence of the last mentioned condition.

The case was manifestly one of primary renal disease with consecutive hypertrophy of the left ventricle, and, later on, fatty degeneration of the hypertrophied heart.

The consecutive change of fatty transformation of the heart was the sole cause of failure of the heart and pulse, and of congestion of the lungs and extremities. It was likewise a contributory cause of the dropsical effusion.

The absence of albuminuria in connexion with cirrhosis of the kidneys, is worthy of special notice.

CASE XXX.—*Albuminuria; Left Hemiplegia, and Mitral Systolic Murmur; Paroxysmal Dyspnœa; Erysipelas; Uræmic Coma; Death. Cirrhosis of the Kidneys, Liver, and Spleen; Hypertrophy of the Left Ventricle, and General Fatty Degeneration of the Heart; Fibrinous Deposit on the Mitral Valve; Dilatation of the Aorta.*

Robert R., aged forty-five years, house-painter; has had a slight attack of painter's colic. Admitted May 4th, 1872. In the course of the preceding summer he suffered from dry retching in the morning and from headache, and shortly afterwards he suddenly lost the use of the left arm and side of the face; but the left leg was affected only in a very slight degree. At the end of six weeks he had so far recovered the use of his arm as to be able to resume his work. In March, 1872, he had a severe

attack of epistaxis; and he noticed, for the first time, that his breathing was affected.

When admitted he was remarkably pallid; face slightly drawn to the right, and on the left side the natural creases were nearly obliterated. The tongue, when protruded, deviated much to the left, and the left arm and leg were weak. Urine albuminous and containing granular tube-casts; œdema of feet; pulse 104, regular; radial arteries rigid and remarkably tortuous, that of the right side making several curves fully an inch in depth. Temporal arteries likewise tortuous and rigid, and in both radial and temporal arteries pulsation was visible. Heart's action weak; apex-pulsation faintly perceptible in nipple-line, and at this point a bellows-murmur of medium intensity accompanied the first sound. At the base the first sound was obscure, and the second sound remarkably sharp and ringing, a character which it preserved throughout the arch of the aorta. Alternations of paroxysmal and suspended breathing; cough and purulent expectoration, and physical evidence of engorgement of right lung. He had several attacks of vomiting during his residence in hospital, and on the 25th May erysipelas of the face, which, under the persistent application of an unguent containing 3j of sulphate of iron to 3j of simple ointment, disappeared in the course of two days.

1st June he became comatose, and on following day he died.

Post mortem, June 3rd. A small quantity of turbid serum was found in the cavity of the peritoneum. The liver was hard and furrowed, and the spleen was in a similar condition, but rather small, the fibrous element being greatly in excess; kidneys contracted and cirrhosed. The lungs were congested, but there was no serum in the cavities of the pleuræ or pericardium. The heart was much enlarged, somewhat flabby, and globular in figure; it weighed twenty-three and a-half ounces. The aorta was dilated and patchy with atheroma; but the lining membrane was natural in colour, and the sigmoid valves healthy and competent. The right ventricle contained a flake of yellow fibrin, which extended in form of a cylinder into the pulmonary artery, half filling the vessel; it was normal as to size and thickness of walls, but its component muscular tissue was in the granu-

lar stage of fatty degeneration; presenting the appearance of lines of translucent dots, and only in one spot in the field, a trace of transverse striation. There was no free fat. (See engraving, Fig XXXI.) The right auricle was likwise filled with decolorized fibrin, which descended into the inferior cava. Left ventricle somewhat dilated, and its walls more than an inch thick in the central portion. The septum projected to the right side. Mitral valve somewhat thick and opaque, but pliant, and competent to close the orifice; some flakes of fibrin were entangled in its edge. The muscular structure of the left ventricle was likewise in a state of fatty degeneration, only somewhat less advanced than that of the right; the transverse striæ being more extensively and more distinctly visible.

Renal disease was likewise the primary affection in this case; and hypertrophy of the left ventricle, fatty transformation of the substance of the heart, and aortic and general arterial atheroma, with dilatation of the aorta, were subsequent changes.

The cause of hemiplegia and of mitral systolic murmur was to be found in thrombosis of the left ventricle, entanglement of a detached flake of fibrin in the margin of the mitral valve, and arterial embolism of the right motor nerve-centre.

The absence of aphasia, in connexion with *left* hemiplegia and the coexistence of rhythmical irregularity of respiration with dilatation of the aorta, without vascular injection of its lining membrane, should be noticed.

Fig XXXI.
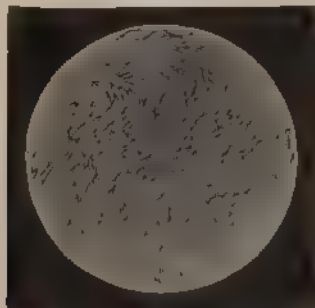
Granular degeneration of muscular substance of right ventricle, × 120. Case of Robert R. This engraving should be examined with a pocket-lens.

CASE XXXL.—*Great Debility and Pallor; Œdema; Visible Pulsation at the wrist; Slight Displacement of the Apex of the Heart to the left; Systolic Apex and Basic Murmur; Systolic Murmur at Ensiform Cartilage; Loud Arterial Murmur; Sudden Death after convulsive seizure. Serous Effusion into Pleura and Pericardium; Fatty Deposition upon, and Granular Degeneration of, Right Ventricle of the Heart; Hypertrophy and Dilatation of Left Ventricle; Flap of Fibrin attached to Ventricular Surface of Mitral Valve and floating into Orifice of Aorta; Dilatation of Tricuspid Orifice.*

Mary D , aged forty-eight years; admitted into hospital June 20th, 1872. Had been short of breath, and generally out of health for some time. Examined on the morning of the 21st, the following was found to be her condition. She was remarkably pale but in no degree cyanosed, and extremely weak; feet much swollen; radial pulse 84, very weak, visible, but regular; urine loaded with lithates, sp. gr. 1 025, and free from albumen. The apex of the heart pulsated slightly to the left of the normal position very feebly, and at this point a loud bellows-murmur accompanied the first sound. At the base a systolic murmur, but more harsh in quality, was likewise audible, and was transmitted thence through the arch of the aorta with great intensity, and also distinctly into the carotids; second sound weak, but normal in both these situations. At the ensiform cartilage the sounds were remarkably sharp and flapping, and here a soft systolic murmur existed.

The chest was resonant in front, and respiration was normal; posteriorly the base was dull on both sides, and the respiratory sounds were feeble.

On the evening of that day (21st) she died suddenly after a convulsive seizure, and on the following morning the body was examined.

There was a good deal of serum in both pleural cavities, and some cretified tubercle in the apex of the left lung, but no adhesion of the pleuræ. There was nearly a pint of serum in the sac of the pericardium, and a considerable deposit of fat on the

surface of the heart, which was slightly in excess of the normal size. The right auricle was dilated; the right ventricle greatly dilated, its walls flabby, and not more than two lines thick in any portion of their extent. In section the outer third was seen to be composed of fat; and the remainder, microscopically examined, was found to be in the granular stage of fatty transformation. The tricuspid orifice was greatly dilated, readily admitting the four fingers and thumb to pass through, and the tricuspid valve somewhat thickened, but pliant and expanded. Both right chambers contained some decolorized fibrin, which extended a short distance into the pulmonary artery. The left auricle was dilated and somewhat thickened. The walls of the left ventricle were greatly thickened, but healthy-looking and firm; its cavity was slightly dilated at the apex, but elsewhere it was of normal dimensions. The anterior segment of the mitral valve was much thickened throughout, irregular at the edge, and incompetent; and on its ventricular surface, about three lines from the free margin, there was attached a tongue-shaped flap of fibrin, about a quarter of an inch in length, and organically united to the valve. When the ventricle was filled with water this flap was floated into the orifice of the aorta, and, during ventricular systole, must have produced by its vibration an eddy in the efflux current, and a systolic basic murmur propagated into the great arteries. The aorta was perfectly healthy; lungs congested posteriorly; other viscera normal.

Mitral valve-disease was in this case the primary affection; giving rise to hypertrophy and dilatation of the left ventricle and auricle, pulmonary congestion, and dilatation of the right ventricle and tricuspid orifice. Fatty deposition upon, and granular degeneration of, the right ventricle, were subsequent changes, and, no doubt, still further impaired its functional capacity, and were the immediate cause of dropsy. The most interesting feature in the case, diagnostically, is that of aortic and carotid murmur due to fibrinous deposit upon the *mitral valve*.

Doctor Flint has recently directed special attention to this phenomenon. I think, however, he over-estimates its frequency. I have no record of a second example of the kind, proved to be such by dissection.

The origin of the epileptiform seizure, which was the immediate cause of death, would probably have been found to be extensive capillary embolism of the brain, had the contents of the cranium been examined, which, I regret, was not the case.

In regard to the relative frequency of hypertrophy from the several causes mentioned, and the average duration of life in each of these categories, the following summary of the preceding fourteen cases may possess some value:

Cause.	No. of Cases.	Average Duration.	Result
Renal disease	7	10 months (nearly)	
Dilatation of aorta	2	24 ,,	
Valvular disease	3	8 ,,	Death.
Rupture of aortic valve	1	8 ,,	
Rupture of mitral valve	1	3 ,,	

It will be observed that the renal form of the malady has furnished quite one-half of the total number of cases given.

It should be noted, however, that this is not due to an absolute preponderance of hypertrophy of renal origin; but rather to the fact, that with this form of the affection, owing to the attendant dropsy, patients are more alarmed about their health and less able to shift for themselves, than they are with hypertrophy from any other cause. They consequently seek hospital relief in greater numbers, and yield a larger comparative mortality.

The duration of life was greatest in those cases in which hypertrophy arose from dilatation of the aorta, and least in those in which it was caused by ruptured valves. This difference was, no doubt, due to the slow progress of atheroma and dilatation of the aorta, during which the left ventricle had time to acquire a proportionate increase of volume and of propulsive power; whereas, in the case of rupture of the valves, the ventricles were surprised, so to speak, and incapable of carrying on the circulation under the increased difficulty suddenly opposed to them.

Dilatation of the chambers of the heart, or "aneurism," as it was designated by Lancisi, was divided by Corvisart into "active" and "passive;" the former corresponding to the "dilatation with hypertrophy" of modern writers, and the latter to "dilatation with thinning" of our nomenclature.

Morgagni gave many examples of both forms. To these Bertin added two others, namely, "simple dilatation," in which the walls are normal as to thickness; and "mixed dilatation," in which portions of the walls are thinned, others thickened, and others again are normal. Bertin held that dilatation of any of the chambers of the heart necessarily implied an obstacle to the circulation in front of it.[*] He thus ignored that form of dilatation, now so common, in which, without obstruction to the circulation, simple debility and softening of the walls of the heart from anæmia or from retrograde changes of tissue, becomes a cause of yielding and dilatation. He commits the further error of assuming that vascular engorgement, passive hæmorrhage, and anasarca, when associated with dilatation, are due to the assumed vascular obstruction which he believes to be a necessary antecedent condition, rather than to dilatation. These consequences ensue, however, only when the heart fails to overcome the obstacle in front, normal or abnormal, and becomes dilated.

The classification adopted by Hope is unexceptionable save as to completeness.[†] He divides dilatation into three forms, namely, "simple dilatation," in which the chamber is dilated whilst the walls are of normal thickness; "dilatation with hypertrophy," the cavity being dilated and the walls thickened; and "dilatation with attenuation," the cavity being dilated and the walls thinned. To these I think the "mixed" form of Bertin should be added; because examples are of not rare occurrence in which one or more portions of the walls, especially of the left ventricle, are normal or actually thickened; whilst at other points, usually near the apex, the wall may be reduced to the utmost tenuity.

Dilatation with comparative rarity engages all the cavities of the heart; and never, save where it is the result of a general or constitutional cause, such as typhoid or fatty softening, anæmia, etc. Hope and Walshe maintain, on the contrary, that dilatation is more frequently general than partial.

But if the frequency with which dilatation, in some degree, of one or more of the cavities is met with, be considered, I am

[*] *Traité des Maladies du Cœur*, p. 382.
[†] *Opus citat.*, p. 294.

convinced the partial or local will be admitted to preponderate numerically over the general form of the affection. If, for example, cases of chronic bronchitis with periodic exacerbations, in which the right ventricle is more or less dilated, and those of primary emphysema of the lungs leading to a similar result, be admitted into the calculation, as they must be; and if the numerous examples of dilatation, in some degree, of the left ventricle and left auricle, in connexion with valvular disease at the mitral or aortic orifice, and accompanied or not by hypertrophy, be added to the preceding list, a moment's reflection will suffice to show that the sum of such cases treated by any hospital physician greatly exceeds the total of those of general dilatation which have come under his personal notice.

Of much greater value than the pathological classification just given, is the clinical division into "primary" and "consecutive" dilatation.

The former comprises cases arising from:

(a) Anæmia.
(b) Over-exertion and malnutrition combined.
(c) Excessive innervation.
(d) Inflammatory, typhoid, or fatty softening.
(e) Excessive use of tobacco.
(f) Onanism.

In the "consecutive" form dilatation may result from:

(a) Obstruction or regurgitation (or both) at the orifice of the aorta.
(b) Obstruction or regurgitation at the mitral orifice.
(c) Atheroma of the aorta.
(d) Obstruction or regurgitation (or both) at the orifice of the pulmonary artery (very rare).
(e) Renal disease.
(f) Obstruction to the pulmonary circulation.

With regard to the mutual relationship of hypertrophy and dilatation, it may be said generally, that when both these states coexist, hypertrophy has been the antecedent, dilatation the consecutive change. Hypertrophy implies increased action of the chamber which has become hypertrophied, and proportionate activity of nutrition; but the causes of hypertrophy, in the

consecutive form at least, are always progressive. Hence, hypertrophy, which is but the measure of resistance to retrograde pressure, is likewise in this form of the affection progressive, so long as the processes of nutritive change in the heart may be quickened. A period, however, ultimately arrives when, although resistance in front continues to increase, the compensating process of hypertrophy of the corresponding chamber of the heart ceases, because interstitial nutrition of its walls fails, either from impairment of the conducting properties of the coronary arteries by atheromatous change of their coats, or from simple starvation of the tissue of a thickened heart by its distance from the nutrient centres, namely, the coronary capillaries. Once this derangement of balance has occurred, and its occurrence is inevitable sooner or later, what Professor Flint appropriately designates the "vital process" of hypertrophy is replaced by the "mechanical process" of dilatation.* Nay more; the form which in any given case a heart undergoing this series of changes actually presents, essentially depends upon the stage of the process at which it is examined, and the stage of thickening at which nutrition of the heart fails. Thus, examined in the early stage, before nutrition had begun to fail, a state of simple hypertrophy, *i.e.*, thickening of the walls, without alteration in the size of the chamber would be found. At a later period thickening may be found still greater, but as yet no change in the capacity of the chamber, a condition differing only in degree from the preceding; or the process of hypertrophy may have ceased owing to early failure of nutrition, and the condition known as moderate hypertrophy with dilatation, or dilatation with hypertrophy, according as one or the other condition preponderated, would be found to exist. In a case where nutrition had progressed to an advanced period, *pari passu* with mechanical resistance to the circulation, and then failed, the heart would be found greatly hypertrophied, with a moderate degree of dilatation; or, at a still later period, disproportionately dilated. The former of these conditions would constitute "hypertrophy with dilatation," and the latter "dilatation with hypertrophy." Simple dilatation and dilatation with thin-

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* *Diseases of the Heart*, second edition, 1870, p. 79, *et sequent.*

ning of the walls are usually primary, although, theoreticall
state of extreme dilatation with or without thinning, may
present the last member of a series of retrogressive changes
heart previously in the highest degree hypertrophied. Su
heart would be greatly enlarged; but on this subject I have
positive information to offer.

I cannot accept the pathological dogma of Rokitansky,*
dilatation is the natural result of valvular regurgitation; whe
hypertrophy follows obstruction. It would be more correc
say that hypertrophy is a *primary*, and dilatation a *second*
result, equally of obstruction and regurgitation. No doubt,
tial dilatation of the left ventricle at the apex may coexist
hypertrophy of the remainder of its walls in permanent pate
of the aortic valve, owing to the long continued pressure wh
it has had to sustain from the reflux column of blood du
diastole. But even here, the dilatation is consecutive to hy
trophy. In what has preceded, it is, of course, implied that
pertrophy can in no instance be secondary to dilatation.

General dilatation of the heart is always of constitutio
origin, and the result of one or other of the causes of " prima
dilatation already mentioned, although these causes may
secondary in operation.† Thus, it may result from anær
and be " primary," in one case; and from the same ca
and yet be "secondary" to valvular lesion and simple hy
trophy in another. Again, it may be due to " primary " fa
degeneration, or to fatty degeneration " consecutive " to
pertrophy from valvular lesion or from chronic renal dise
The distinction is important; because, as I will hereafter sh
the latter or secondary form of general dilatation, is by m

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* *Pathological Anatomy*, vol. iv., p. 162.

† Doctor Stokes many years ago foreshadowed these views, in the following p
graph, which came under my notice only when the preceding passage had
written. I need not say how much pleasure it afforded me to find my views
tained, because anticipated, by the great authority of my illustrious teacher.
is still to be determined whether the cavities of the heart are liable to change
mechanical causes alone ; whether dilatation, for example, is a purely mecha
result of obstruction to the exit of the blood, or whether for its productio
valvular disease there is required not only obstruction, but a weakened condi
of the heart." (*The Diseases of the Heart and Aorta*, 1854, p. 255.)

the more serious prognostically, and the more rapidly fatal of the two.

Beau, indeed, maintains that general dilatation with hypertrophy of the heart is the legitimate pathological offspring of pericarditis issuing in universal adhesion of the pericardium; the false membranes, by their contraction, exercising a general dilating force upon the heart.* In reference to this theory, regarding which Beau declares that " he cannot adopt it in an absolute manner," it may be remarked, that where the pericardium is not extensively adherent to the chest-wall, there is no sufficient *point d'appui* whence the false membranes can exercise a general excentric traction upon the heart. All such cases I believe to be examples of previous hypertrophy, from whatever cause, with subsequent pericarditis and inflammatory softening and dilatation of the heart.

The *symptoms* and *signs* of general dilatation of the heart will be found to vary within certain limits, according to the stage at which it is examined. Thus, at an early period of its history, yet when the characteristic features are sufficiently pronounced, the patient will exhibit languor of mind and body; the extremities, ordinarily of a slightly livid tint, will be readily chilled and benumbed; the pulse, small and regular, may intermit at long intervals; respiration is normally shallow, and readily quickened by exertion, and the patient is easily put out of breath by physical effort, or mental excitement. There is a constant hemming or teasing cough, accompanied by scanty serous expectoration and a proclivity to catching cold, which constitutes the patient's principal infirmity. On these occasions there is great embarrassment of breathing, which may amount to orthopnœa; the pulse is quick, feeble, and it may be irregular; the conjunctivæ injected with dark blood, and the features generally livid and somewhat puffed.

In the ordinary condition of the patient secretion and excretion are performed with regularity, although the liver is somewhat engorged, and the patient may be troubled with hæmorrhoids. The appetite, however, is indifferent, and digestion is slow; and from time to time, as shown by foul tongue

* *Archives Générales de Médecine*, vol. x., April, 1836.

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eructation and anorexia, the stomach suffers derangeme
function, either as a consequence of some slight indiscreti
regard to food, or in damp and foggy weather without
such provocation. On such occasions the urine precipita
copious sediment of lithates. The extent of precordial du
is horizontally increased, if not masked by emphysem
lungs. The impulse of the heart is feeble, diffused, and
sionally intermittent; and a distinct point of apex-pulsatior
rarely be detected. The precordial region, viewed in pi
will generally exhibit indistinct fluctuation in one or mor
tercostal spaces. The sounds of the heart are sharp and dis
the first closely resembling the second in brevity and pitch
varying as to clearness and intensity according to the sta
the cardiac walls; it is feeble and low toned, though wel
fined, when the walls are fatty or otherwise softened, but
and ringing when tissue-change does not exist. The puls
of the heart is quickened by even the slightest movement o
body, and on such occasions it is attended with throbbing o
carotid arteries, and visible fluctuation of the external ju
veins.

In a few such cases, as I have elsewhere shown,[*] the
sound at the apex is accompanied or succeeded by a soft bell
murmur, due, as I believe, to inadequacy without disease of
mitral valve. If, at the acme of systole, a portion of the
of the ventricle to which one or more of the papillary mu
are attached, yielding under the centrifugal blood-pressure o
to its softened or attenuated condition, is carried outwards a
angle from the axis of the ventricle, it is readily conceivable
one of the flaps of the mitral valve may be thereby lifted of
opening through the medium of the tendinous chords, an
give rise to mitral reflux and murmur. Under treatment,
murmur may cease coincidently with the occurrence of g
ral improvement in the condition of the patient (see Case
and 37).

Doctor Walshe would regard regurgitation in such case
due to general dilatation of the left chambers and mitral or

[*] *British Medical Journal,* December 18th, 1867.

and consequent inadequacy of the valves ; but manifestly, if the auriculo-ventricular ring were dilated, it would, owing to its tendinous structure, be incapable of recovering its former dimensions under tonic treatment, so as to adapt itself to the inadequate valve and prevent further reflux. Yet, if we will not admit that the valve itself may have undergone expansion to the requisite extent to fill the opening, and that this process may have been, in a series of cases, coincident with treatment rather than consequent upon it (for the treatment that has been successful in removing the murmur could have no effect in expanding the valve), an assumption which I am by no means prepared to grant, we shall be forced to believe that such changes of expansion and contraction of the tendinous ring have actually taken place. I repeat that, in my opinion, such cases are explained by the existence of localized softening, or extreme attenuation of the wall of the left ventricle.

When general dilatation has attained its maximum, respiration is shallow, laboured, and protracted, and usually accompanied by a loud wheeze ; there is complete orthopnœa, and impending suffocation when the patient assumes the recumbent posture even for a moment. The features, especially the lips, tip of the nose, and lobes of the ears, are livid, the conjunctivæ congested and somewhat jaundiced, and the eyelids and face generally bloated ; surface and breath cold ; the feet cold, puffed, and congested. The pulse is slow, feeble, intermittent, and irregular. Precordial dulness extended horizontally, but difficult to be defined with accuracy, because the anterior borders of the lung, which are usually emphysematous, overlie the precordium to some extent. The heart pulsates at the ensiform cartilage ; its action is feeble, flapping, irregular, and barely perceptible to the hand ; and if it be not entirely overlain by the edges of the lungs, a fluctuating movement of its anterior wall is perceptible in one or more of the intercostal spaces, at the end of expiration. The sounds are faint, but sharply defined and high pitched, and if the walls be thinned but not softened, the first sound so closely resembles the second in quality as to be distinguishable from it only by its rhythm, as determined by the cardiac impulse or carotid pulsation. Fine crepitant râles are audible

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over the bases of the lungs, which are somewhat dull, and
chest generally is either non-vibratile or very feebly so. Th
is constant teasing cough, accompanied by thin mucous
frothy expectoration frequently tinged with blood.

Anasarca, and serous effusion into the cavities of the chest
abdomen quickly supervene, the latter being usually the im
diate cause of death. In such case the mode of death is by co
resulting from the combined operation of retained renal and ;
monary excreta. Another mode of death is, however, not un
quently exemplified in this affection; namely, that by thromb
of the right side of the heart and pulmonary artery.

The conditions upon which this accident, for as such it i
be regarded, depends, are partial stasis of the blood by fail
of contraction in the right chambers of the heart, and by
pairment of respiratory and nutritive attraction arising f
feeble respiration and arrested tissue-change.

The symptoms which indicate its occurrence are, a sense
precordial oppression, gradually increasing to a point of gi
intensity, and attended with tumultuous but feeble action of
heart; great dyspnoea and craving for air; restlessness and si
ing; the patient starts from his slumber with great alarm, and
entirely deprived of refreshing sleep; the pulse is quick, we
irregular, and fluttering, or all but entirely suppressed at .
wrist; the surface is cold, damp, and livid; respiration is ra
full, and laboured, but manifestly ineffectual, although air fre
enters the lungs, and loud rhonchi or wheezing sounds are he
all over the chest. The carotid arteries pulsate with great v
lence, and the jugular veins are turgid and fluctuating. T
most characteristic symptom of this condition is, however,
besoin de respirer. The arms are thrown out and extended;
covering is flung off the chest; the patient begs that fresh
may be admitted, and declares he is being suffocated, althou
the chest moves freely and extensively, and there is abund
circulation of air in the chamber, and, as judged by auscultat
evidence, in the lungs also. In this condition death usua
occurs quite suddenly on the patient's making the slightest
ertion, such as that of sitting up; it is the immediate con
quence of the impaction of a mass of fibrin in the tricus

orifice or the pulmonary artery, by which the circulation in the lungs is mechanically arrested, the systemic arteries emptied, and the veins gorged with blood. General dilatation of the heart is least promising where the organ has undergone change of structure; it is of unfavourable augury in proportion to the extent and degree of tissue change which the heart has suffered. That of fatty degeneration is the most serious, because the least amenable to treatment; it is likewise the most liable to terminate in sudden death by thrombosis or rupture.

That form of the affection which is secondary to hypertrophy from valvular lesion, or from renal disease, is likewise proportionately more serious than that of primary origin, because of the retrogressive tissue-change which it implies, and the formidable mechanical resistance which, in either case, the heart, thus weakened by tissue-decay and by thinning of its walls, has to overcome in carrying on the circulation.

General dilatation from anæmia, or from excessive labour and inadequate nutrition, is, on the other hand, the most promising form of the affection, and susceptible not only of improvement, but of cure under appropriate and persistent treatment. Dilatation from "typhoid softening" is usually met with in typhus fever, or consecutive to it, and is likewise amenable to treatment.

I have met with only a few, certainly not half a dozen, examples of it in the typical form described by Dr Stokes, although it must have been not rare at the time he wrote.

Treatment.—In the anæmic form, iron is, *par excellence*, the remedy. It should be given in such form as the stomach can best tolerate, and its efficacy will be greatly enhanced if it be combined with strychnia and quinine, as in the syrup of the triple phosphate, of which a teaspoonful may be given in twice that quantity of cold water thrice daily. Where dilatation is the result of excessive physical toil, worry, and malnutrition, as so frequently exemplified in poor women of weak frame and nervous temperament, digitalis and iron combined act as a specific (*vide* Case 37, Mrs. C.) I usually prescribe ℥ v–x of the tincture of digitalis with ℥ x of the tincture of perchloride of iron, and ℥ v of chloric ether, in an ounce of water every third hour.

Nutritious diet, including a large proportion of animal food and a liberal allowance of wine, are at the same time ordered. The bowels, which are usually constipated, but may be over-relaxed, should be attended to. In the former contingency, grs. v of the compound aloetic pill, with a-quarter gr. of extract of nux vomica, may be given every night; and in the latter, ℞x of dilute sulphuric acid, or of the liquor of pernitrate of iron, in an ounce of water, thrice or oftener daily.

In that form of dilatation which results from softening, whether inflammatory, typhoid, or fatty, I rely chiefly upon quinia and strychnia combined; ℞v of the liquor strychniæ of the Pharmacopœia, with grs. iss to ij of quinia, in an ounce of water, acidulated with dilute sulphuric acid and sweetened to the taste, should be given every third or fourth hour. Nutritious diet and an ample allowance of wine should be provided; moderate exercise in the open air should be taken daily; but the ascent of steeps, and indeed all causes of excitement of the circulation, whether mental or bodily, and of fatigue, should be studiously avoided.

Doctor Stokes was the first to point out the great benefit to be obtained from the use of mercury in the treatment of fatty and dilated heart, with engorgement of that organ, the liver and lungs, paroxysmal dyspnœa, and anasarca. There seems some reason to conclude that these attacks are associated with latent gout. Dr. Stokes gives, in illustration, the case of the late eminent surgeon Abraham Colles, who ultimately died of this affection. Writing of Mr. Colles, he says: "He continued to suffer from time to time, from paroxysms of dyspnœa, which were generally preceded by diminution in the secretion of the kidneys. During these attacks, which generally lasted for several days, the irregularity of the heart and the precordial distress increased until orthopnœa was established. The kidneys acted scantily, and no copious sediment appeared in the urine. On each attack, the tumefaction of the liver increased with great rapidity; but this condition as rapidly subsided with the improvement in the symptoms. No relief was ever obtained until a free action of the kidneys was established; but it was found that this could only be effected by the use of mercury, followed by diuretics.

On several occasions the diuretic treatment, not preceded by mercury, was tried; but it always failed, so that the number of times in which a distinct course of mercury was employed, was very great. To this remedy, in a great degree, must the prolongation of Mr. Colles' life be attributed, for, on various occasions, the symptoms had gone so far as to cause complete orthopnœa, with unusual anasarca, and alarming pulmonary congestion."[*]

I have no personal experience of the use of mercury in cases such as that described by Dr. Stokes; but were an example of the kind placed under my care, I should have no hesitation in adopting the plan of treatment so successfully followed by him.

I have repeatedly observed temporary irregularity in the action of a dilated heart to be directly due to derangement of stomach, as manifested by flatulence, acid eructations, and foul tongue. Dr. Stokes has likewise noticed this, and truly remarked that the cardiac irregularity may be promptly corrected by measures directed to the gastric derangement. I think cases of this kind are also associated with the gouty diathesis, and under the use of acetate of potash and tinct. of colchicum, grs. xxx of the former with ℳx of the latter, given every third hour in an ounce of orange-flower water, and preceded by a saline purgative, I have found the regularity of the heart's action restored in the course of twenty-four to forty-eight hours.

For the relief of precordial oppression arising from excessive cardiac distention, the application of two or three leeches over the base of the heart is the measure most likely to be efficacious for the time. I have repeatedly seen patients who had been entirely deprived of sleep for several nights in succession by this feeling of oppression and impending suffocation, enjoy tranquil and refreshing sleep for several hours after the application of two leeches to the precordium.

In the treatment of the alarming condition due to thrombosis of the heart, in addition to rest, warm applications to the feet, and wine or spirits in small and oft repeated doses, I would make trial of the plan recommended by Dr. B. W. Richardson,[†]

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[*] *The Diseases of the Heart and the Aorta*, p. 297.
[†] See *Medical Press and Circular*, of November 20th, 1872.

although I have had no actual experience of it; namely, tha[
giving ℞x of the spirit of ammonia in an ounce of water e[
hour, and grs. v of the iodide of potassium, every alternate h[

The treatment of dilatation associated with persistent palp[
tion of the heart and enlargement of the thyroid body, with[
without prominence of the eye-balls, constituting the mo[
condition known as Graves' disease, and affording a typical[
ample of dilatation from excessive innervation, should be [
ducted mainly with reference to the general condition of [
patient. That condition is preeminently one of debility, [
most frequently of anæmia likewise. Hence the indication[
alcoholic stimulants in moderate quantity, iron and digitalis, [
regimen of a bracing character. I usually allow four to [
ounces of sherry daily, and prescribe tincture of the perchlo[
of iron with tincture of digitalis, of each, ℞x thrice daily, i[
ounce of water. For the relief of intercurrent precordial dist[
I direct two leeches to the precordium, and afterwards an op[
plaster. In cases of extreme nervous susceptibility I have gi[
half drachm doses of bromide of potassium. In all cases I [
commend cold shower baths and moderate exercise out of do[
and, when it can be afforded, the use of chalybeate waters [
some months in a bracing climate, such as Harrogate.

Dilatation resulting from the excessive use of tobacco is ch[
acterized, most frequently, by slight intermittence of the pu[
and a tendency to syncope; likewise by a fugacious syst[
murmur at the apex of the heart, the action of which is usu[
quick, but variable as to rate independently of physical ex[
tion. The patient is pale, fidgetty, and generally "nervous," [
he gives, when questioned, the history of excessive indulgenc[
tobacco-smoking. It is, in many cases, idle to recommend a t[
discontinuance of the practice of smoking. The wiser cours[
to urge upon the patient gradually increased restriction in [
respect, and the use of the mildest "smoking-mixture" obta[
able. In addition, cold shower baths should be recommend[
and, medicinally, strychnia and iron. For example, ℞v of [
liquor strychniæ, with gr.iss of the granulated sulphate of iro[
an ounce of water thrice daily.

Onanism, and the cardiac debility and dilatation which re[

from it, and which are characterized, as in the preceding example, by nervous excitability and palpitation, and not unfrequently by systolic apex-murmur without valvular disease, are best treated by absolute prohibition of the vicious habit to which they are due ; and this should be enforced, if necessary, by vesication of the genitals, or by some equivalent expedient. Bromide of potassium in full doses, 3ss to 3j, is, *par excellence*, the medicinal agent most to be relied upon. It should be given in the above mentioned quantity, thrice daily, for several weeks consecutively, with occasional intervals of one or two days, for the purpose of avoiding the general depression and nausea likely to arise from its long continued use.

Dilatation arising from any of the preceding causes, though general, may affect the two sides of the heart or the individual chambers unequally. Thus, fatty softening is essentially ventricular, rarely engaging the auricles in a great degree, and usually affecting the ventricles very unequally. Typhoid softening likewise most frequently affects the left ventricle in a greater degree than the right, and the consecutive dilatation exhibits in either case a corresponding extent and rate of progress.

Dilatation from nervous debility and habitual excitement, as exemplified in Graves' disease, always exhibits a preponderance in the left, as compared with the right ventricle.

Consecutive dilatation, or that arising from obstruction or from a local, as distinguished from a general, cause, is nearly always secondary to hypertrophy, as previously shown. It occasionally happens, however, that the process of atheromatous change in the orifices and valves of the heart, or in the aorta, commences simultaneously and proceeds concurrently with that of fatty degeneration of the substance of the heart. In such a contingency dilatation of the chamber undergoing retrogressive change of its walls, provided the obstruction lay directly in front of it, would be " primary." But that this is of exceedingly rare occurrence may be inferred from the rarity of dilatation without thickening of the walls, in connexion with atheroma of the valves or of the aorta.

Consecutive dilatation, as depending on a local cause, is itself most frequently local in the first instance. It may, however,

affect both sides of the heart at the same time; but in such a
two concurrent causes, one affecting each side, must be in o
tion: a contingency exceedingly rare. Not so, however, th
retrogressive dilatation when the disease commences on th
side. In this case, owing to obstruction to the pulmonary
culation, the right side of the heart would be affected su
sively with hypertrophy and dilatation.

When dilatation of the right ventricle is the result of pri
obstruction in the lungs, as when it follows emphysema wit
repeated intercurrent bronchitis, the retrogressive proces
arrested in the systemic veins, and never, in my opinion
tends beyond the capillaries to the left side of the hear
have not met with a single example which would constitut
exception to this statement. Pulmonary obstruction, to an
tent and degree adequate to the production of general ve
engorgement, when not rapidly fatal, is always intercurrent
temporary.

The systemic veins being remote from the seat of obstruc
and not having suffered permanent impairment of tonicit
temporary distention, readily recover their normal calibre v
free circulation through the right side of the heart and the l
is restored; and the left heart, thus relieved from backv
pressure through the capillaries, will not have undergone
the temporary obstruction thence arising, any alteration eithe
regard to the thickness of its walls, or the capacity of its cl
bers. Not so, however, the right side of the heart, which, ov
to its proximity to the seat of obstruction in the lungs, and
feebleness of its walls, is found, after repeated distention
have become permanently dilated.

The rule may be therefore regarded as general, that w
obstruction on the left side of the heart is competent to pro
hypertrophy and dilatation of the right side; primary stasi
the right side is not reciprocally productive of hypertrophy
dilatation on the left side.

Dilatation of the right chambers of the heart in its *pri*
form may be due to:

(a) Fatty deposit.

(b) Fatty degeneration.

(*c*) Inflammatory softening.

(*d*) Fibroid conversion.

In the *secondary* form it may result from :

(*a*) Obstruction in the lungs.

(*b*) Obstruction or regurgitation at the mitral orifice.

(*c*) Obstruction or regurgitation at the aortic orifice.

In the primary form, dilatation of the right ventricle is usually associated with thinning of its walls; but when secondary to any of the causes indicated in the latter category, it is preceded by hypertrophy, provided that general nutrition be unimpaired; hence there is thickening of the walls with enlargement of the cavity. But, as already stated, hypertrophy of the right ventricle, even in a moderate degree, is rare.

The differential diagnosis, from direct physical evidence, of dilatation of the right ventricle, accompanied by, or unattended with, thickening of its walls, is not practicable in the present state of medical knowledge ; but dilatation of some form having been determined, the history and collateral signs actually existing will enable the physician to form a presumptive judgment almost equivalent to a positive diagnosis.

This difficulty arises from the twofold circumstance, that the right ventricle is shielded from the touch by the sternum, and that the factor contributed by it to the first sound is so insignificant, that even when exaggerated, it is not distinctly appreciable.

Dilatation of the right ventricle can scarcely be presumed to exist without a corresponding state of the right auricle, owing to the invariable accompaniment of expansion of the tricuspid orifice. The right auricle may, however, be dilated without accompanying dilatation of the right ventricle ; when, for example, organic obstruction exists at the tricuspid opening, without a corresponding condition of the mitral orifice, a very rare occurrence, the right auricle would alone undergo hypertrophy and dilatation.

Dilatation of the right ventricle may be diagnosed by extension of precordial dulness to the right, unaccompanied by displacement of the apex to the left side, and by the existence of jugular pulsation synchronous with ventricular systole. This

latter sign, first pointed out by Lancisi, but by him, and
sequently by Laennec, erroneously interpreted as eviden
hypertrophy of the right ventricle, is really diagnostic
of dilatation of the right ventricle, but of tricuspid inade
as shown by Morgagni. This latter condition is, how
rarely met with, except as the result of dilatation of the
ventricle, which may be, therefore, likewise presumed to
Hope regarded this sign as evidence of hypertrophy with d
tion of the right ventricle; but Bertin correctly interpreted
a sign of dilatation only of that chamber, with a correspo
condition of the right auriculo-ventricular opening, and c
quent inadequacy of the tricuspid valve.

Morgagni drew a just distinction between jugular puls
resulting from simple auricular reflux, and that from ventr
regurgitation. In the former case, as he remarks, the puls
of the jugular vein observes no relationship in time to the
tole of the ventricle; whereas, in the latter, it is strictly
chronous with the pulse, and therefore with the contracti
the ventricle.*

This distinction is, as I believe, well founded. A trem
movement of the jugular veins, extending over the entire per
ventricular diastole, and corresponding to the feeble undul
contractions of the auricle prior to its final and abrupt presy
contraction, is of frequent occurrence, and represents a r
degree of congestion of the right side of the heart. I have,
ever, repeatedly observed both kinds of jugular movement i
same case; viz., a full reflux wave synchronous with the cor
tion of the ventricle, and a feeble and continuous move
immediately succeeding it: both being readily arrested by
finger-pressure above the clavicle.

Longet, adopting the views of Potain, represents the ju
veins as normally exhibiting the following series of moveme
viz., two abrupt elevations in quick succession; the former
to the arrest or retardation of the entrance of blood into
auricle at the moment of its contraction, and the latter, t
retrograde shock or pressure arising from the closure of the
cuspid valve, or the lateral pressure of the great arteries

* *Seats and Causes of Disease*, translated by Cooke, 1822, vol. i., p. 377

the adjacent veins at the moment of their expansion; perhaps to both these causes combined. Alternately with the twofold expansion of the vein just described, may be noticed a partial subsidence or sinking of it, due to the successive diastole of the auricle and the ventricle; and, finally, a slow movement of expansion from the refilling of the vein from the periphery.[*]

The annexed figures (XXXII. and XXXIII.) of Friedreich, copied from Dr. Gee's work,[†] represent abnormal venous pulsation. Figure XXXII. shows *systolic* dicrotism of the jugular

Fig. XXXII

Fig. XXXIII.
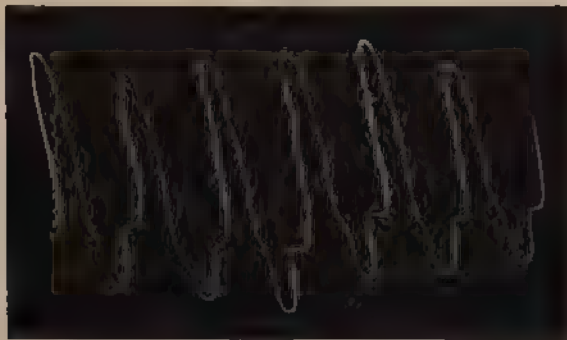
[*] *Traité de Physiologie,* 1869, tom. deuxième, p. 250.
[†] *Auscultation and Percussion,* 1870, p. 167.

pulse, the first and lesser portion of the wave of ascent bei
result of auricular contraction, the second and larger, th
ventricular systole. Figure XXXIII. represents a phenor
of occasional occurrence, namely, *diastolic* dicrotism of the
lar pulse. This is the result of persistent repletion of the
auricle, and consequent sudden arrest, towards the end of
tole, of the further entrance of blood.

Dilatation with thinning of the right ventricle, from soft
whether fatty or inflammatory; and dilatation with hypert
from emphysema and recurrent bronchitis, or other chron
struction to the pulmonary circulation, are of less unfavor
augury than the corresponding changes in the left vent
because of the much less liability to rupture of a softened
ventricle than of a left ventricle similarly affected, and be
chronic obstruction to the circulation in the lungs is usua
a remittent character. There is, however, considerable d
from thrombosis of the right ventricle and pulmonary arter

Dilatation of the right auricle is usually associated with
consecutive to dilatation of the right ventricle, owing t
facility with which, in protracted engorgement of the right
tricle, the tricuspid orifice becomes expanded, and its
thereby inadequate. It may exist, however, as already s
independently of any change in the capacity of the right
tricle, where tricuspid narrowing is the only valvular lesior
sent; but this is a pathological unity of extreme rarity,
separate dilatation of the right auricle is, therefore, very se
witnessed.

A right auricle dilated in a high degree may simulate
rism of the ascending aorta. The most remarkable examy
this kind on record is given by Dr. Stokes. A man, past m
age, was admitted into the Meath Hospital suffering from s
toms of cardiac disease. The heart pulsated in its natural
tion; its action was feeble and irregular. There was dulne
the right of the sternum, extending from the second to the
rib, and to an equal extent in this situation an expansile p
tion was felt resembling that of an aneurism, and synchro
with the first sound of the heart. No note was taken
whether murmur existed. In regard to aneurism, a neg

diagnosis was made, chiefly from the coexistence of irregular action of the heart. The patient sank from congestion of the lungs. The aorta was healthy; the lungs were emphysematous and congested. The right ventricle was dilated and hypertrophied, and the right auricle "resembled a vast purple tumor which concealed the whole of the anterior portion of the right lung. Its parietes were in many places extremely thin, while in others the fleshy columns, especially in the appendix, were hypertrophied. Its cavity contained more than a pound of fluid but grumous blood."* The pulsation of the auricle was here due, as Dr. Stokes remarks, to systolic reflux from the ventricle. The difficulty of differential diagnosis from aneurism must have been very considerable in this case, and in the enumeration of possible sources of error, the example, though unique, should not be lost sight of. The necessary coexistence, however, of pulmonary and general venous engorgement must have aided considerably towards a right conclusion.

Where the right side of the heart is dilated and engorged, whether from debility of the walls or from primary obstruction in the lungs, I would not hesitate to abstract blood locally, or even generally, but with circumspection. The application of a few leeches to the precordium, or the abstraction of a few ounces of blood by cupping over the lungs where they are the seat of primary obstruction, the use of the hot foot-bath and a mild saline aperient, are usually followed by great relief to the patient. Where, in the absence of valvular disease on the left side, general venous congestion amounts to a state of cyanosis, with great dyspnœa, distention of the cervical veins, irregular pulse, and feeble, but tumultuous action of the heart, I would not hesitate to bleed from the arm to ten or twelve ounces. Medicinally, I consider digitalis, when the stomach will tolerate it, the most valuable agent we possess in the treatment of weak and engorged right heart. I prescribe it in doses of ℞ x of the tincture, with an equal quantity of chloric ether and of tincture of perchloride of iron, in an ounce of water every third hour. I have never witnessed from its use more unpleasant results than

* *The Diseases of the Heart*, p. 274-5.

occasional sickness of stomach; but when this occurs the medicine should be stopped for at least twenty-four hours. It increases the contractile force of the heart, slowing its action, and restoring its regularity of function. Thus, by its action, when it is administered in well selected cases, the strength and volume of the radial pulse are increased, and its regularity, if absent, is restored, whilst the renal secretion is augmented. The allowance of liquids should be very limited, and alcoholic stimulants should be given in full quantity.

Dilatation of the left, like that of the right auricle, when it exists separately, depends upon contraction of the auriculoventricular opening of that side, and is associated with thickening of its walls. It must, however, ultimately give rise to dilatation of the right chambers, and should, therefore, be regarded as representing an early stage of a process which must issue in more general dilatation of the heart.

From the anatomical position of the left auricle, dilatation of this chamber must be difficult of recognition by independent signs; but, in conjunction with those of mitral narrowing, the existence of continuous feeble undulatory pulsation in the second and third intercostal spaces of the left side, about one inch from the sternum, may be regarded as evidence of this condition.

Separate dilatation of the left, like that of the right ventricle, may be primary, and dependent upon typhoid, inflammatory, or fatty softening; or it may be secondary to organic disease at the mitral orifice, involving valvular inadequacy and regurgitation, or to disease at the orifice of the aorta giving rise to obstruction, regurgitation, or both, provided there be softening of the walls.

Softening of whatever kind, if primary, is followed by dilatation with thinning of the walls, when time has elapsed sufficient for the complete development of the pathological series.

The symptoms are weak and failing pulse, which is, likewise, usually quick, intermittent, and irregular. It may, however, be abnormally slow and large in the fatty form of softening, but here the radial artery is usually tortuous, and not unfrequently rigid. Vertigo is of frequent occurrence, and there is a tendency to syncope on suddenly assuming the erect posture, and on

making unusual effort. In cases of softened and dilated left ventricle, the apex-point, as determined by the sounds, is displaced to the left, and the apex-beat is feeble and diffused. The first sound is weak, but short and high pitched; the impulse is either in the last degree feeble, or altogether inappreciable. At the base the second sound is sharp, but weak, and if the aorta be not rigid by atheromatous or calcareous change, it is but faintly audible at the right second costal cartilage. In the treatment of typhoid softening, as pointed out by Dr. Stokes, wine in full quantity constitutes the remedy; under its use the pulse usually becomes slow at the same time that it acquires volume and strength. Alcohol is also indispensable in the other two forms of softening of the left ventricle, and in all three I prescribe quinine, strychnine, and iron.

When dilatation of the left ventricle is the result of valvular lesion it is always accompanied by thickening of the walls. The diagnosis and the treatment will be considered in connexion with the valvular lesion upon which the dilatation depends.

The following are examples of dilatation of the heart.

CASE XXXII.—*Dilatation of the Heart and Resolution of the First Sound.*

Catherine M., aged fifty-five years, a domestic servant, came under my notice as an extern patient of the hospital, March 12th, 1869. Had been for some time previously suffering from flatulence and sensations of "sinking" at the stomach, vertigo, and occasional faintness. Had never spat blood. Was pale and thin, but free from cough; pulse 96, weak, and occasionally intermittent. Sounds of heart morbidly clear over precordium generally; but, at the apex, the first sound was resolved into its two elements of impulsion and valve-click, of which the former was dull and soft, whilst the latter, which immediately succeeded, was sharp and "valvular" in character. The former, by its rhythm, might be mistaken for a presystolic murmur, but was readily distinguishable from it by the soft character of the sound, and by its coincidence with the cardiac impulse. R. Tincture of digitalis and chloric ether, of each ℥ v thrice daily. Not seen again.

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Although I incline to associate reduplication or resolution of the first sound at the apex with simple hypertrophy of the left ventricle as the usual attendant condition, I would not be understood to deny its occasional occurrence in connexion with other organic changes of the heart; for example, thickening, with some degree of dilatation, of the left ventricle.

CASE XXXIII.—*Dilatation and rapid Action of the Heart; Irregularity and want of Correspondence between the Action of the Heart and the Radial Pulse.*

Margaret L., a widow, aged forty-five years, visited the hospital as an out-patient in February, 1868. Twelve years previously she had swelling of the legs after cold caught in her first confinement. She is remarkably nervous and fretful; pulse at wrist 108, so weak as to be with difficulty counted, and arythmically irregular and intermittent; heart acting at rate of 192, irregular, but not intermittent, its sounds sharp and clear, and accompanied by a musical note not in harmony with either sound; apex-beat half an inch external to the nipple. Feels weak occasionally after an effort, and is sometimes faintish after rising from bed, so that she is obliged to lie down again, but has never fainted; breathing on these occasions much accelerated. To have aperient pills; also tincture of iron and chloric ether, of each ℥ij, with infusion of quassia to ℥viij: a tablespoonful to be taken thrice daily.

Admitted into hospital, April 27th. Heart acting at rate of 182; sounds clear and ringing, but unaccompanied by murmur. Pulse weak, irregular, and intermittent, and, as to rate, out of harmony with the action of the heart. To have tincture of digitalis, tincture of the American wild cherry (Prunus Virginiana), and chloric ether, of each ℳv, every fourth hour.

May 7th. Pulse and heart-rate correspond, 96 per minute with slight irregularity. No sensation of sinking; can sleep and take food well; heart-sounds less clear and sharp. Discharged.

June 17th. Pulse weak, irregular, and intermittent, 168, and corresponding with action of heart. To resume last medicine.

April 2nd, 1869. She again presented herself. Pulse 96,

with an uncertain number of abortive beats; action of heart varies from 144 to 168; sounds clear. There are, occasionally, a few strong and regular beats; these are represented in the radial pulse, and are succeeded by an uncertain number of weak pulsations in rapid succession, which are not fully, or only by abortive pulsations, represented in the radial pulse. She suffers from dizziness, and is in danger of falling after exertion, especially that of ascending a flight of stairs.

The musical note audible at the precordium on the occasion of the first visit, but not subsequently, was of gastric origin. I have repeatedly heard this sound in connexion with gastric tympany, and have had no difficulty in identifying its source by its metallic character; its want of *strict* synchronism with either sound; its capriciousness, or occasional absence during one or more cycles of cardiac movement without any intelligible cause; and, finally, by the existence in the precordium of tympanitic percussion-resonance continuous with that of the epigastrium.

The beneficial action of digitalis is exemplified in the comparative regularity, increased force, and reduced rate of the heart's action, and in the general improvement of the patient under its use in May, 1868.

CASE XXXIV.—*Hypertrophied, Dilated, and Fatty Heart; "Ascending and Descending" Respiration; Sudden Death.*

Joseph C., aged sixty-two, an educated man of gigantic stature, presented himself as an extern patient, May 12th, 1869. He stated that during the preceding fifteen years he had suffered from shortness of breath, with occasional attacks of bronchitis, and that a fortnight previously he had spat some blood. There was no œdema; the pulse was 96, arythmically irregular, and intermitting. He was subject to sudden accessions of dyspnœa, with tendency to fainting, which usually came on in bed, and obliged him to rise promptly in order to catch his breath. Had not been able to lie down for the preceding week, owing to the immediate accession of attacks of this kind on his attempting to do so. To have tincture of digitalis, tincture of perchloride of iron, and chloric ether, of each ten drops, thrice daily.

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May 14th. Pulse 96, intermitting; feet swollen; heart's action irregular and intermittent; its sounds clear, but weak, and free from murmur. Respiration "ascending and descending" at intervals, and then his sight is dim. Declares he feels better, and has been able to get some sleep since he began to take the medicine.

19th. Admitted into hospital. There is now great oedema of the lower extremities, and dyspnoea threatening asphyxia; pulse intermitting; breathing "ascending and descending," or exhibiting alternations of gradual increase and decrease of rate, succeeded by a brief period of complete apnoea. To have previous medicine.

20th. Slept well in recumbent posture last night, and declares he feels infinitely better.

23. No return of dyspnoea. Sleeps well; oedema disappearing; attributes his improvement to the medicine, which he calls his "preservative," and demands to have it repeated. Passed since yesterday about a gallon of pale urine.

This man was discharged at his own request a few days afterwards, feeling well and free from dyspnoea, the oedema having entirely disappeared. The pulse, however, continued irregular.

July 16th. Called on me at my residence to-day. Had an aggravated attack of dyspnoea last night, causing him to start out of bed to save himself from suffocation. Is very thin ; pulse irregular and failing; great oedema of feet and legs; kidneys and bowels acting well. No cardiac impulse to be felt, but a general and feeble movement is perceptible to the hand placed on the precordium. At the apex and in the line of the aorta both sounds were remarkably obscure, whilst at the ensiform cartilage they were morbidly clear. No haemoptysis. To have, thrice daily, ℳvj of the tinctures of digitalis and iron, with chloric ether, as previously.

17th. Visited at his lodgings. Passed a better night, and reported much improved.

August 8th. I discontinued my attendance, the patient being in all respects improved, able to sleep in the recumbent posture, appetite fairly good, oedema having disappeared ; but the action and sounds of the heart and the radial pulse still were as above noted.

A few weeks subsequently to the last mentioned date, I learned, through the kindness of Dr. Dudley White, surgeon to the city coroner, that this man had died suddenly at his lodgings. Dr. White examined the body at the coroner's inquest, and informed me that the heart was large and fatty; but no more special report was made.

The advantage derived from the use of digitalis, even where the heart is fatty, is here strikingly exemplified.

CASE XXXV.—*Dilated and Fatty Heart.*

Patrick D., aged sixty years, a labourer, temperate, and never had rheumatism, was admitted into hospital, June 12th, 1868. Nine weeks previously his breathing became short after a wetting, and a month later his feet began to swell. Respiration 36; pulse 130, and irregular; tongue coated, bowels constipated and flatulent; well marked arcus senilis.

The urine was acid, sp. gr. 1.025, and free from albumen; feet œdematous and congested. Muco-crepitus with inspiration over apex of left lung, and here respiration was feeble, but elsewhere it was normal; slight jugular pulsation of inspiratory rhythm; precordial dulness normal; both sounds of heart sharp and clear at the apex and at the base; action of heart irregular, but no cardiac murmur to be heard.

June 15th. Pulse 120, irregular; great increase of urine.

16th. Pulse 84, irregular; heart-sounds sharp and clear; urine, about three quarts daily. Discharged improved, and not subsequently heard of.

CASE XXXVI.—*Weak and Dilated Left Ventricle in a gouty subject; Postsystolic Apex-murmur; Cessation of all Morbid Signs under Treatment.*

Mr. W., aged fifty-six years, unmarried, and temperate, holding a responsible position in the Civil Service, requiring nine hours of office duty daily, consulted me, March 15th, 1872. Five years previously, after a fainting fit in church, he consulted an eminent physician, whose opinion, as reported by him, was that he had valvular disease of the heart. Another physician of no less eminence, subsequently expressed a similar opinion.

He was well nourished, could take exercise with enjoyment, and felt himself improved by it; had no cough or derangement of respiration, and no œdema; had never spat blood. Pulse 96, small, regular, and equal, but occasionally intermitting; apex-beat in nipple-line and fifth intercostal space, and of moderate force. Both sounds were here distinct and normal; the first, however, was sharper than natural, and was succeeded by a faint blowing murmur (postsystolic), extending quite up to the second sound, but inaudible elsewhere than at the apex. Both sounds were a little more sharp than normal at the base, and the second sound was not accentuated in the pulmonary artery,

This gentleman had had rheumatic fever in childhood; his habits were irregular in regard to diet, no food whatever having been usually taken between breakfast at 9, A.M., and dinner at half-past 6, P.M. He declared that he had been rather improving in health during the last five years, and he desired to have a definite opinion as to the state of his heart, with a view to claiming a retiring allowance under the regulations of his department.

I gave a confident opinion that no valvular disease existed; that the symptoms and signs referable to the heart were due to weakness and dilatation exclusively; and that, under treatment they would entirely disappear. I added, that his illness was, in my judgment, connected with latent gout. To have ℳv of tincture of digitalis, with ℳx of tincture of perchloride of iron, and ℳiij of tincture of colchicum and spirit of chloroform thrice daily. To have every night, with a view to correction of flatulency with which he was much troubled, gr. j of blue pill, with gr. j of compound rhubarb pill, and grs. ij of dried soda. He was directed to take one or two glasses of good dry sherry daily, and to have some luncheon at mid-day.

I have since repeatedly seen this gentleman; he had continued the use of the medicine above mentioned for several months, and was when I last examined him (September, 1872) in excellent health, and quite free from all morbid signs referable to the heart. The murmur which existed at the apex on the occasion of the first visit, ceased within a few weeks.

It would be presumption in me to attempt to explain why the two deservedly eminent men who had diagnosed valvular lesion,

had arrived at that conclusion. I may, however, be pardoned the suspicion that the error into which they undoubtedly fell, was due to the existence of a systolic murmur, having its origin at the apex, and associated with rheumatic antecedents. A mitral murmur of systolic or postsystolic rhythm, is even still, by most stethoscopists, regarded as *prima facie* evidence of organic lesion of the mitral valve. In a previous Chapter (page 282), I have endeavoured to show that reflux murmur at the mitral orifice may exist as a result of weakness and excentric yielding of a portion of the wall of the left ventricle, at the acme of systole, and without structural alteration of the mitral valve. I believe the case now under consideration was an example of this kind ; and the cessation of the murmur coincidently with the general improvement of the patient under tonic treatment, may, I think, be legitimately offered as evidence of the correctness of that opinion.

CASE XXXVII.—*Weak and Dilated Left Ventricle ; Non-organic Mitral Murmur.*

Frances C., aged fifty-eight years, the mother of eight grown children, became an out-patient of the hospital in the autumn of 1865. She had worked hard in early life, and twenty years prior to her visit, she began to suffer from weakness and palpitation on making unusual exertion ; latterly she had been subject to intercurrent attacks of bronchitis. In the spring of 1867 she felt more than ordinarily weak, and experienced lightness in the head after quick movement or exertion of any kind. In the course of the preceding winter she had a slight attack of rheumatism, in which the right elbow, knee, and ankle were swollen, but there was no complaint of pain in the left side during this attack. Pulse 84, and in an extreme degree arythmically irregular ; it likewise occasionally intermitted twice or thrice in succession, sometimes only once. The radial arteries were tortuous, and pulsated visibly. Respiration regular, 30 in the minute, and unembarrassed. No venous turgescence of the neck, or visible pulsation of the carotid or temporal arteries. Precordial dulness normal in extent. Cardiac impulse weak, and apex-pulsation slightly to left of usual position. The first

sound was accompanied by a faint murmur at the apex, and
second, at midsternum, was likewise accompanied by a feeble
mur, which seemed to constitute the initial portion of the so
the terminal portion of which was normal. Distinguished
murmur, the sounds were remarkably sharp and clear. To
clean, bowels confined. To have aperient pills, and amn
and chloric ether in camphor water, also a moderate allow
of wine. She was subsequently directed to take $\mathfrak{m}$ v of li
strychniæ, in a bitter infusion, thrice daily.

July 26th. Reported that she can now rise at 5 o'clock,
a feat previously impossible. Pulse still irregular and 84,
occasionally slower, owing to the occurrence at intervals o
abortive pulsation not represented in the radial pulse. *No
diac murmur now to be heard.* Medicine continued.

November 19th. Feels weak, action of heart and radial p
arythmically irregular and intermitting; heart-sounds clear,
no cardiac murmur.

April 5th, 1869. Pulse 96, still occasionally irregular;
feels very well, and is able to work.

June 12th. Slight œdema of face; action of heart and p
arythmically irregular and intermitting; looks well, but c
plains of uneasy sensations in region of heart, with a dist
perception of its intermissions. To have tincture of the
chloride of iron, chloric ether, and tincture of digitalis, of e
$\mathfrak{m}$ x in an ounce of water, thrice daily. Having, at this t
ceased to conduct the extern department of the hospital, I
sight of this patient, but I have since repeatedly seen her in
streets, and to all appearance in her usual health.

The diagnosis of mitral reflux by yielding of the walls of
already weakened and dilated left ventricle, and without val
lar lesion, was made at the first interview with the patient
1867. The subsequent cessation of the murmur under t
treatment showed that this view of its origin was correct.
occurrence, at one period, of a faint murmur at the orifice of
aorta, prefacing the second sound, subsequently ceasing, and
returning, is explicable only on the assumption of a shre
fibrin attached for the time to the free edge of the valve,
rendering it partially inadequate at the moment of closure.

Atrophy of the heart must be regarded as the opposite of its enlargement. Under the various forms of hypertrophy and dilatation, all possible conditions of the organ, in regard to thickening of its walls and dilatation of its chambers, have been considered. Most, if not all these conditions imply enlargement of the heart, the only *theoretical* exception being dilatation with thinning of the walls ;* but, even here, I am convinced there is absolute enlargement in nearly every instance, from the preponderance of dilatation over parietal thinning.

Bouillaud understands by atrophy the opposite of hypertrophy, and recognizes three forms of it corresponding to the three forms of the opposite condition, hypertrophy; viz., *simple atrophy*, in which the walls are attenuated and the cavities normal as to capacity ; *excentric atrophy*, or attenuation of the walls, with dilatation of the cavities ; and *concentric atrophy*, the walls being normal or increased in thickness, and the cavities reduced in capacity.

Bertin makes a similar classification, but under different designations. The heart must be, however, in every instance below the normal standard as to volume, according to him.

Having reference to the general reduction in volume of the heart necessary to constitute a state of atrophy, I think a preferable, because a more natural, classification would be the following ; *simple atrophy*, consisting in a general reduction in volume of the heart, the walls and cavities bearing a normal proportion to one another and to the size of the organ ; *excentric atrophy*, in which, whilst the heart is below the normal size, the walls are thinned and the cavities dilated relatively to the walls ; *concentric atrophy*, in which, the heart being in volume less than normal, the walls are relatively thickened and the cavities reduced in capacity.

Rokitansky enumerates the following causes of atrophy of the heart, viz., general marasmus from typhus, tuberculosis, or the cancerous diathesis ; the pressure of secondary products in the mediastinum ; pericarditis ; fatty accumulations on the heart ; and contraction of the openings of the coronary arteries. The

* I purposely omit so called " concentric " hypertrophy, the existence of which, as a pathological condition, I cannot recognize.

tissue of the organ may, he says, be either reddish-brown
tough, or fawn-coloured and easily torn.* As stated by Be
atrophy of the heart is due to diseases other than cardiac;
there is at least one exception, namely, where atrophy depe
upon adhesion of the pericardium.

Doctor Stokes is of opinion that atrophy, quite as often as
pertrophy, is found associated with adhesion of the pericardi
and he surmises that the former condition is due to pressure up
and obliteration more or less complete of, the coronary arteries
the contraction and pressure of the false membrane. He furt
states, on the authority of Professor R. W. Smith, that calcif
tion of the pericardium is, when present, invariably associa
with atrophy of the heart.†

Barlow‡ and Cheevers§ have maintained that obliteratior
the pericardium by adhesion is more frequently followed
atrophy than by hypertrophy of the heart.‖

Doctor Walshe, whilst holding that hypertrophy is the o:
nary change of the heart consecutive to adhesion of the p
cardium, admits that he has met with at least one exampl
atrophy of the left ventricle from the constricting action of
enveloping false membrane. He attributes the wasting in
instance to pressure upon the coronary artery.¶

The most frequent causes of atrophy of the heart are undou
edly diathetic diseases of debility, and, *par excellence*, tuber
losis and cancer, as first pointed out by Louis and Bizot.
heart in such case is pale, but firm, and structurally sou
except in so far as it may be the seat of tubercular, cancer
or other adventitious deposit; and its walls and cavities ar
normal relative proportion.

I have not seen atrophy from accumulation of fat upon
heart, and pressure therefrom on the coronary arteries, as m
tioned by Flint.** By atrophy I understand reduction in

* *Pathological Anatomy*, Sydenham Society's edition, vol. iv., p. 170.
† *Opus citat.*, p. 12.
‡ *Gulstonian Lectures*, 1843.
§ *Guy's Hospital Reports*, vol. ix.
‖ *Vide antea*, p. 347.
¶ *The Diseases of the Heart*, p. 274.
** *Opus citat.*, second edition, 1870, p. 92.

total volume of the heart. In the form mentioned, however, there is no diminution of volume, the proper substance of the heart is only in part replaced by an adventitious structure. Atrophy of the heart may be due to atheromatous change in the coronary arteries, or to a similar affection of the aorta obstructing the entrance to these vessels. In such case it would be associated with retrogressive change of structure, granular or fatty, and the heart would be likewise soft and lacerable, and of a dark fawn or a light brick-colour. It may be likewise congenital, the viscera generally, and the body itself being similarly undeveloped. The heart, therefore, would be in due proportion to the remainder of the body, and its walls and cavities strictly proportionate. Many hold that atrophy of the heart may be the result of general senile decay; but Cruveillier denies atrophy of the organic muscles in the aged, whilst admitting that of the muscles of animal life.* In this opinion I entirely concur. Wasting of the organs essential to life, in the absence of specific disease, is not an ordinary accompaniment of advancing years. In acute muscular atrophy, the voluntary muscles are usually alone affected.

There is, however, at least one case on record, in which the heart participated in the wasting and degeneration of this disease. The patient was a female, aged twenty-six years, under the care of Dr. Headlam Greenhow. She died of wasting palsy, which was characterized by the usual symptoms, within a period of less than five weeks. The muscles of the forearms, the erectors of the spine, the pectorals, and the diaphragm, were wasted, and had undergone the diffuse granular change usually exhibited by certain of the voluntary muscles in this disease. The heart was pale and somewhat fatty on the surface, and its muscular structure, especially that of the right ventricle, exhibited well pronounced granular degeneration, with some interfibrillary fat.†

Chronic pericarditis, with general adhesion of the pericardium, may be the cause of atrophy of the heart. I have already ex-

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* *Anatomie Pathologique,* l. xxx.
† *Clinical Society's Transactions,* vol. vi., 1873.

pressed the opinion (p. 368) that hypertrophy is the ordinary result of pericarditis with adhesion, occurring at a period of life when general nutrition is active, *i.e.*, in childhood and youth, provided the health of the patient be otherwise good, and no special impediment to the nutrition of the heart exists, such as obstruction of the coronary arteries. In the opposite circumstances, that is, where pericarditis occurs in a person of feeble nutrition, whether from advanced age or broken health, or where it is preceded or accompanied by obstructed coronary circulation, the result is likely to be atrophy, and most probably that of the degenerative kind.

Chronic effusion into the left pleural cavity may, by the pressure exercised upon the heart, and the general impairment of nutrition to which it usually gives rise, be the cause of atrophy of the heart. Dr. Flint gives an example of this kind in an adult female. The heart was flabby, fawn-coloured, and probably fatty; it weighed only four and a-quarter ounces, and the walls of the left ventricle were less than a-quarter of an inch thick.[*]

The "pigmentary atrophy" of Reynaud[†] is usually met with in combination with fatty degeneration, and is, I believe, a form of that change, but modified by the accident of pigmentation. In this condition the section of the heart exhibits a bronzed or slate colour, and in the fibres pigment granules are observed in addition to oil dots.

The pulse in all cases of atrophy of the heart is small, quick, and, it may be, all but inappreciable; but, in those forms in which it is unassociated with fatty or other tissue-change in an advanced stage, it is regular. In the latter, however, the pulse is irregular, intermittent, and failing, and syncope is of frequent occurrence. The area of precordial dulness, both deep and superficial, is contracted, and the cardiac impulse is limited and feeble. The sounds are sharp and clear where the structure of the heart is sound; but where it is softened, the first sound is dull, faint, and scarcely audible. Flint is certainly in error when he states that the sounds are "abnormally feeble or inappreciable," irrespec-

* *Opus citat.*, p. 93.
† *Nouveau Dictionnaire de Médecine et Chirurgie*, 1868.

tively of the structural condition of the heart. In infants, with still smaller hearts than those met with in most examples of atrophy, the sounds are remarkably clear and distinct, even *in utero.*

Atrophy will be exemplified in the following cases.

CASE XXXVIII.—*Atrophy with General Dilatation of the Heart.*

Bridget M., aged sixty-six years, pale and delicate looking, visited the Mater Misericordiæ Hospital as an out-patient in 1867. She was then suffering from palpitation, the action of the heart and the radial pulse being arythmically irregular and intermittent; there was also a tendency to syncope, and partial but temporary failure of vision. Both sounds of the heart were equally clear, sharp, and distinct. She was relieved by the use of iron, digitalis, and chloric ether. She again presented herself in May, 1870, complaining of a hard and painful tumor in the left axilla, which was manifestly malignant; she then suffered also from great weakness and from the former tendency to syncope. The cardiac rhythm was deranged, the impulse being both arythmically irregular and unequal. Thus, there would be two or three beats in rapid succession, but at unequal intervals, and at a rate of more than 100 in the minute, and then a long intermission both in the impulse of the heart and in the radial pulse would occur. The sounds of the heart were very clear and sharp. I lost sight of her after this date.

CASE XXXIX.—*Hepatic Symptoms with Dropsical Effusion into the Peritoneum ; Variation in the Volume of the Liver, and Enlargement of the Spleen ; Paracentesis Abdominis, and Peritonitis ; Irregularity of the Heart's Action, and Muffled First Sound ; Anasarca and Hydrothorax ; Paracentesis Thoracis ; Death. Chronic Pericarditis with General Dense Adhesion and Calcification ; Excentric Atrophy of the Heart ; Hydrothorax with Carnification of the Lungs ; Cirrhosis of the Liver ; Enlargement of the Spleen and Kidneys ; Chronic Peritonitis.*

James R., a young man of good education, aged nineteen; was

admitted into the Mater Misericordiæ Hospital in June, 1863, suffering from uneasy sensations, rather than pain, in the region of the liver. There was ascites, and likewise œdema of the left lower extremities; pulse small, weak, and variable as to rate, but regular. Paracentesis abdominis was performed, and nine gallons of serum were removed. Two months subsequently he had improved so much in health that he was able to leave hospital. There was then no ascites, and no enlargement of the liver could be detected.

Five months later the liver was found enlarged, and irregular on the surface, and there was slight effusion of serum into the peritoneum. The pulse at this date was full, strong, and regular. General health good.

One month subsequently there was jaundice which lasted two days; and two months later still, viz., in May, 1864, he had so much improved in health that he had gained flesh. Pulse 102, and full; liver much enlarged and thickened, but smooth on the surface.

In August there was again liquid accumulation, but only to a slight amount, in the abdomen; the liver was tender to pressure, and the left leg was œdematous.

From this date till April, 1868, when he was readmitted into hospital, the patient presented himself at intervals of a few months. It is unnecessary here to give details as to his varying condition during this lengthened period. These I have elsewhere published at length.* I shall give such portions only as are relevant to the present subject.

On the 31st May, 1866, a note was for the first time made that the action of the heart intermitted.

On the 13th July, 1867, more than a year later, the further observation was recorded, that "The first sound of the heart was masked, the second sound being exceedingly clear, and the heart still irregular in action, and intermitting."

Under date 15th November following, it is noted that "both sounds were remarkably clear, and of a ringing metallic character."

There were recurrent attacks of jaundice, which usually lasted

* *Proceedings of the Pathological Society of Dublin*, vol. iii., part i., 1866, p. 281.

not more than a few days; the spleen became perceptibly enlarged; serous effusion now took place into the cavities of the right pleura and the pericardium, but very slowly, and the quantity present varied from month to month, inversely as the action of the kidneys. The ascites, however, steadily increased, and, concurrently, the patient lost flesh and became weaker; the lower extremities and genitals became so swollen that it was found necessary to puncture them in order to avoid gangrene, and enable the patient to micturate.

On the 19th March, 1868, both sounds of the heart, as heard beneath the lower sternum, were clear and ringing as before, but the action was irregular and intermitting; urine, sp. gr 1·018, acid, and containing lithates and a trace of albumen.

On the 25th April the right pleural cavity was full of liquid, the cervical veins were engorged, the face was livid, and respiration was so much embarrassed that death from asphyxia seemed imminent. Paracentesis thoracis was performed, and shortly afterwards the patient sank exhausted.

Both lungs were found congested, and the base of the right carnified. The abdominal cavity was full of dark serum; the viscera were universally adherent by dense false membrane, which enveloped the liver in a thick envelope. In the recesses between the agglutinated intestines small pools of serum were found, which glistened with crystals of cholesterine. The liver was in an advanced state of cirrhotic and fatty change. The spleen and kidneys were greatly enlarged and congested, but structurally healthy.

The pericardium was calcified, greatly thickened, and closely and firmly adherent to the heart. It presented a cartilaginous appearance in most parts; but in certain situations it was calcified, and resisted the knife; and in a few places on the right side, and to a less extent on the posterior left aspect of the left ventricle, there were large masses of gritty calcareous matter, partly imbedded in the pericardium, and partly in the substance of the heart.

The vena cava superior and the vena azygos were considerably dilated, showing the obstruction to which the abdominal venous circulation had been for a long time subjected. The

heart was much reduced in size, soft, and flabby. The right cavities were dilated, and their walls remarkably attenuated. The tricuspid orifice was large, and both auricle and ventricle were occupied by masses of decolorized fibrin, connected through the tricuspid opening and extending into the pulmonary artery. The left auricle was greatly dilated, and the left ventricle was dilated, and its walls soft and greatly thinned. There was no valvular disease whatever. The substance of the heart was of a dark fawn colour, but not fatty.

It is not improbable that at the date of the first observation of intermittence of the heart, viz., in May, 1866, latent pericarditis existed; and in July of the following year the muffled character of the first sound may have been due to liquid effusion into the pericardium, which was subsequently absorbed, and followed by adhesion and calcareous conversion of the adventitious matter.

I conclude that in the foregoing case atrophy of the heart was due to the twofold cause of general malnutrition and special interference with the nutrition of the heart, arising from the close adhesion of the pericardium. Calcification of the adventitious connective tissue was a subsequent event, and likewise an example of atrophic degeneration with deposition of calcareous particles.

CASE XL.—*Scirrhus of the Mesentery; Death by Peritonitis from Sloughing of the Intestines and Effusion of their Contents. General Adhesion and Calcification of the Pericardium; Simple Atrophy and Granular Degeneration of the Heart.*

A poor man named P., a native of North Wales, aged forty-three years, was admitted into hospital on the 26th November, 1872, on the recommendation of my friend Dr. Roberts of Portmadoc. He had complained for six months previously of occasional dull pain in the abdomen, and irregularity of the bowels, and, when admitted, he was much emaciated. He suffered from dull remittent pain in the hypogastrium, which was aggravated whenever the bowels were confined; a hard, rugged, and movable tumor was detected in the right iliac fossa; there was

occasional diarrhœa, the stools being free from fæcal odour, and resembling liquid mortar. The pulse was quick and small, but regular, precordial dulness limited in extent, and cardiac impulse remarkably feeble; the sounds were weak but clear, and unattended with murmur. A few days before his death, which took place on the 31st December, the abdomen became inflated and tender, and owing to this twofold cause the tumor became imperceptible to the touch.

Post mortem. Sloughing and perforation of the lower portion of the ileum had taken place, and peritonitis had been set up. A large mass of scirrhus, readily broken up, and partly detached, and stained by the effused fæces, occupied the folds of the mesentery adjacent to the cæcum. A small scirrhous nodule was found in the walls of the cystic duct, obstructing the passage. The gall bladder was distended with bile. No scirrhus existed elsewhere in the abdomen or in the chest.

The heart was reduced in volume; it weighed only six ounces. The muscular substance was of a dun-brown colour, firm to the touch, and in the granular stage of degeneration; the cavities and walls were relatively normal; the great vessels and the coronary arteries were reduced in size proportionately to the heart, but they were otherwise unaffected. The pericardium was universally and firmly attached to the heart, and corrugated; and the adventitious connecting medium was the seat, near the base of the heart, of several plates of osteoid formation, and of two so called cartilaginous laminæ. The latter, examined microscopically, were found to consist of nucleated fibroid tissue, resembling non striated muscular fibre after treatment with acetic acid. No cartilage corpuscles or lacunæ existed.

Atrophy of the heart in this case was of later date than adhesion of the pericardium, as shown by the corrugated state of the latter, and was a consequence of the general failure of nutrition arising from the cancerous cachexy. As in the preceding case, the formation of osteoid and chondroid substance, in connexion with the products of antecedent pericarditis, was the result of failure of nutrition, and, in my opinion, coeval with atrophy of the heart.

CASE XLI.—*Leucocythæmia. Cirrhosis of the Liver, and Concentric Atrophy of the Heart.*

A gentleman, who had been a veterinary surgeon in the army, and had served in India for seven years, came under my notice professionally on the 18th September, 1862. He had been invalided, and after his return from India, had undergone a course of mercury for secondary syphilis. He was of very delicate frame, pale, nervous, and emaciated. The appetite was capricious, the bowels irregular, and sleep was obtained only by the free use of opium. The spleen was manifestly enlarged; but no other organic disease could be detected. He continued under my treatment for nine months. During that time he suffered from slight neuralgic pains in various parts of the body, from periostitis of the tibia and of the cranium, and he had an attack of testitis. The urine frequently exhibited a copious precipitate of lithate of ammonia. In the course of the winter, cachectic ulcers, apparently syphilitic, appeared upon the legs, and the feet became swollen. He was frequently unable to take food of any kind.

In the succeeding summer, under the advice of an eminent physician, he visited Barége and drank the waters; but, after six months' residence there, he returned in no degree improved. Whilst at Barége he suffered from continual diarrhœa, which he attributed to the use of the waters; the ulcers upon the legs, however, had healed.

I saw him again in September, 1864, when, in addition to anasarca, there was ascites; and on the 23rd November he became a private patient of the Mater Misericordiæ Hospital. He now began to suffer much from flatulent distention of the large intestine. A drop of blood, obtained from his finger by pricking with a needle, exhibited an excess of white corpuscles, which almost equalled the red corpuscles in number. The ascites increased rapidly, and his debility became extreme. The dejecta from the bowels, which were constipated, were of a light colour, and exceedingly fetid; there was no trace of jaundice.

He died, worn out, on the 21st December.

At the examination of the body, the left pleural cavity was

found nearly full of serum, and the lower portion of the left lung compressed. The right lung, which was emphysematous, presented five lobes instead of three. The heart was very small, not exceeding that of a child of five years; the right cavities were in all respects normal, proportionately to the size of the heart; but the left ventricle was in the condition of *concentric atrophy*, its walls being much thickened, and its cavity reduced in size. The substance of the heart and its valves was structurally sound.

There was a large collection of serum in the peritoneum, and the spleen was greatly enlarged. The liver was in an early stage of cirrhosis, and the gall bladder was distended with bile. The kidneys were somewhat enlarged, and exhibited a few patches of fatty degeneration. Attached by a fine pedicle to the posterior false ligaments of the bladder, were four, small, round bodies, of a yellow colour and smooth surface, consisting of a cortex, which was composed of small cells, and an enclosed plexus of capillary blood-vessels. For a more full report of this case, the *Proceedings of the Dublin Pathological Society*, New Series, Vol. ii., Part ii., 1864, may be consulted.

It is scarcely necessary to remark that the foregoing case illustrates atrophy of the heart produced by a general or constitutional cause of a non-malignant character; namely, the peculiar and probably specific blood-crasis, known as leucocythæmia.

The three forms of atrophy of the heart mentioned at the outset, will be found exemplified in the preceding cases.

I shall now proceed to the consideration of the special or *heteroplastic diseases* of the substance of the heart.

These may be discussed under the following heads, viz. :

  1. Fatty disease.
  2. Fibroid.
  3. Cancerous.
  4. Syphilitic.
  5. Hydatid.
  6. Tubercle.

In the discussion of these various subjects the above order will not be strictly observed.

Fatty disease of the heart, as conventionally interpreted, includes two conditions of that organ which differ widely in regard to their genesis, the anatomical lesion involved, the degree of impairment of function, and the benefit to be expected from treatment. These are, *fatty deposition or infiltration; and fatty degeneration.*

Fatty deposition or growth is simply an extension into the domain of disease of the physiological process of " growing fat." It consists in the absorption by cells of free fat from the blood. The connective tissue corpuscles of the sub-cutaneous areolar tissue, of that of the orbit, mesentery, etc., perform this process normally; and abnormally, the hepatic cells, and the corpuscles of the perimysium. It occurs more readily when there is excess of free oil in the blood.

From the mode of its production, fatty infiltration of the heart must be located externally to the sarcolemma of the muscular fibres; and in this situation it is exhibited in the form of fat cells of average size and configuration. In both these respects fatty infiltration differs anatomically from fatty metamorphosis, as will be presently shown.

In fatty infiltration, function is not abolished by destruction of tissue; it is only mechanically impeded by encumbrance and by partial absorption of the muscular fibre; and, finally, it is much less grave prospectively, and more amenable to treatment than fatty metamorphosis.[*]

It may be conveniently divided into *superficial and interstitial* infiltration of the heart, as proposed by Quain. Of these, the former is normal, and the latter abnormal, anatomically considered.

Fatty metamorphosis,[†] *or degeneration* of cellular structures, consists, according to Rindfleisch, in a decomposition of the amalgam of albumen and fat, of which the cells are composed, the fat being set free, and in muscle collecting in the form of

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[*] *Rindfleisch Pathological Histology*, New Sydenham Society's edition, 1872, vol. i.

[†] Fatty metamorphosis, as employed by Rokitansky, means deposition of fat cells *between* the muscular fibres; not transformation of the sarcous elements into oil-clots, as implied in the text.

groups of oil dots in the interfibrillary protoplasm of the fibres. This aggregation may be observed especially around the nuclei, owing to the large size of the spaces formed here for their reception by divarication of the adjacent fibrillæ.[*]

Hoppe-Seyler, Burdach, Kemmerich, Voit, and Ssubotin have shown that fat may be the product of the retrograde metamorphosis of albuminoid bodies.[†]

Fatty metamorphosis of cells occurs normally in the epithelium; for example, in that of the lactiferous and the sebaceous glands. But when it occurs pathologically it is due to a derangement of proportion between the means of nutrition and the structure to be nourished; and such disproportion may result either from a diminution of the *nutriens*, or an increase of the *nutriendum*.[‡]

Defect of pabulum may arise from a local or from a general cause. As examples of the former may be adduced, obstruction of the coronary arteries by atheromatous or calcareous change in their coats, or by fibrinous thrombosis or embolism,[§] and the disturbance of circulation arising from the inflammatory process.

In parts recently inflamed there is an inadequate supply of pabulum, not only from the attendant disturbance of circulation, but likewise from mechanical arrest to the transit of nutrient material by massive proliferation of corpuscular elements. To the derangement caused by the defective supply of nutriment thence arising, may be added that resulting from increase of parenchyma by inflammatory products.

Fatty degeneration of the heart may likewise arise from general failure of nutrition (and this is its most frequent cause), whether from absolute want of the elements of a wholesome and sufficient dietary, as amongst the poor; from failure of tissue-assimilation; or from failure of circulation through athero-

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[*] *Antea*, p. 596.

[†] Doctor Macalister, in *Dublin Quarterly Journal*, August, 1870.

[‡] Rindfleisch, *opus citat.*

[§] It is generally held that the tissue-change which takes place in the heart, consecutive to thrombosis or embolism of the coronary arteries, being the result of an acute cause, is softening rather than fatty degeneration.

matous change of the arterial coats. To these changes gouty subjects of middle or advanced age are especially liable; hence the frequent association of gout, arterial atheroma, and fatty degeneration of the heart. Whether imperfect disintegration of albuminous substances in the liver is the cause of lithæmia, gout, and atheroma, as Dr. Murchison, with good reason, suspects to be the case,* it is certain that atheromatous degeneration of the aorta and coronary arteries is the most frequent precursor of fatty degeneration of the heart.

Poisoning by phosphorus has been also an occasional cause of fatty degeneration of the heart and other viscera. Several examples of this kind may be found in the works of Orfila and Taylor; and a very striking example has been published by Dr. Habershon.† A woman, aged twenty-eight, took by mistake a quantity of rat poison, containing about five grains of phosphorus. She exhibited the usual symptoms of phosphorus-poisoning, and died on the fifth day after the occurrence. The liver, spleen, kidneys, and heart, as well as the voluntary muscles, were in a state of fatty degeneration. In the "Year-Book" of the New Sydenham Society for 1860, a case, published by Lewinsky, is referred to, in which death followed the taking of a large dose of phosphorus, and the liver was found to be " lardaceous."

In the volume of the same publication for 1863, another case of the same kind is quoted from the writings of Bucquoy, in which the brain was found to have undergone fatty degeneration.

When due to obstruction of the coronary artery, or to a general cause, fatty metamorphosis would be " primary;" and when dependent upon preceding inflammation, it would be " secondary," as classified by Quain. In this latter category I would include fatty transformation of the substance of the heart, supervening upon, or secondary to, hypertrophy.

Doctor Ponfick proposes the division of fatty degeneration of the heart into the " senile" or " plethoric;" and the " anæmic." The subjects of the former are aged and fat, the arteries are degenerate, and the costal and tracheal cartilages are calcified.

* Croonian Lectures, *British Medical Journal*, 1874.
† *Medico-Chirurgical Transactions*, vol. l., 1867.

The fibres of the heart frequently exhibit pigmentary as well as fatty change.

The subjects of the second, or anæmic form, are young and anæmiated. There is slight fatty change of the internal and middle coats of the arteries, defect of red blood corpuscles and fibrin, and relative excess of white corpuscles. He adds, that in the former cases the fatty change is usually local, and in the latter it is general.[*]

Quain notices the frequent association of recurrent hæmorrhage with fatty degeneration of the heart in young persons, and Virchow states that fatty degeneration of the heart is a common accompaniment of chlorosis.[†]

Having thus shown that there are two distinct forms of fatty disease of the heart, and defined briefly their genesis and anatomical differences, I shall proceed, in the next place, to give a succinct but necessarily imperfect history of the subject. For the materials of this sketch, prior to 1850, I am mainly indebted to the able and exhaustive memoir of Dr. Quain.[‡]

It is necessary, however, to mention at the outset, that owing to the disadvantage of a total want of acquaintance with, or an imperfect appreciation of, the distinction between fatty growth and fatty degeneration, under which the early writers on this subject were placed, a certain amount of confusion and obscurity was unavoidable in their description of symptoms and anatomical lesions. Hence, it will be necessary, in the classification of symptoms which have been assigned to fatty disease of the heart generally, to distinguish carefully between those which are proper to the one and to the other form, respectively.

Hippocrates remarks, that "persons who are naturally very fat are apt to die earlier than those who are slender;"[§] and Celsus, that "whilst thin persons are feeble, fat persons are unenduring."[‖]

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[*] *London Medical Record*, February 19th, 1873, quoted from *Berliner Klinische Wochenschrift*, 1873.

[†] *Ibid.*, January 8th, 1873.

[‡] *Medico-Chirurgical Transactions*, vol. xxxiii.

[§] Works translated by Adams, *Aphorism* 44.

[‖] "Gracile corpus infirmum ; obesum hebes est ;" *De Re Medica*, liber ii., sec. i.

Galen alludes merely to the inconvenience of corpulence, and to the necessity and the means for reducing it.[*]

Harvey describes the heart of old Parr, who died in 1635, as laden with fat.[†]

Lancisi likewise alludes to fatty accumulation upon the heart.[‡] And Morgagni gives some examples of this condition, of which the following deserves special notice on account of the symptom of fainting exhibited.

A very fat woman, aged seventy-five years, subject to fainting fits ("*quasi-deliquia*") died suddenly. The mediastinum and the heart were loaded with fat, and death had occurred by rupture of the left ventricle on its posterior surface.[§]

Corvisart had never seen an example of fatty change of the fibres of the heart, but alludes to it as having been witnessed by others. He had, however, frequently met with examples of fatty accumulation upon the heart, and admits that this condition may be the cause of sudden death. [||]

It would appear, therefore, that to Corvisart, although he had very little personal experience of the disease, is due the merit of having been the first to recognize the distinction between the two forms of fatty change of the heart.

Laennec, whilst describing minutely the different forms of "softening of the heart,"[¶] does not even allude to accumulation of fat upon it, or to fatty transformation of its substance.

Doctor A. Duncan junior, in 1816, published the case of a female, aged twenty-two years, who died rather suddenly after severe and protracted pain in the region of the heart. The pericardium was found universally adherent, and the substance of the heart, to the depth of two-thirds of its thickness, replaced by fat.[**]

In 1818 Cheyne published his well known case, which merits special notice in a historic summary of this subject, because cer-

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[*] *Methodus Medendi*, liber xiv., c. xv.

[†] Works translated by Willis, Sydenham Society, p. 590.

[‡] *De Motu Cordis, Romæ*, 1728, p. 55.

[§] *De Sedibus et Causis Morborum*, epistola xxvii., observation 2.

[||] *The Diseases of the Heart*, Hebb's translation, pp. 166 and 168.

[¶] *Auscultation Médiate*, tom. ii., p. 533.

[**] *Edinburgh Medical and Surgical Journal*, 1816.

tain specific symptoms are here for the first time noticed in connexion with a fatty condition of the heart.

A full, stout, and florid man, aged sixty years, addicted to the pleasures of the table, gouty, and subject to occasional attacks of catarrh, exhibited for the last few years of his life œdema about the ankles in the evening, and occasional intermittence of the pulse.

After a long and fatiguing walk he had fluttering and palpitation of the heart, followed by a "fit," which was believed to be apoplectic, in which he lost consciousness; and the pulse became irregular and unequal. For this, which was followed by a sense of fulness and pain in the head, he was bled to nearly twenty-eight ounces within a period of little more than an hour; and shortly afterwards he had fifteen leeches applied to the head.

Under this treatment he recovered, save as to the pulse, which continued irregular and unequal. The feet and legs soon afterwards became swollen, and he had a slight attack of articular gout. He survived the fit about two months, and for the last few days of his life the respiration was irregular; it would cease for a-quarter of a minute, then be feebly resumed, increase gradually in rate and energy, and then gradually subside till it was again suspended. In Cheyne's words: "For several days his breathing was irregular; it would entirely cease for a-quarter of a minute; then it would become perceptible, though very slow; then, by degrees, it became heaving and quick; and then it would gradually cease again. This revolution in the state of the breathing occupied about a minute, during which there were about thirty acts of respiration." Shortly before death he became hemiplegic (right) and aphasic. The brain was found congested over the left hemisphere. Two ounces of serum had collected in the pericardium. The heart was three times its natural size, and the right ventricle was entirely converted into fat in its lower part, and very thin superiorly. The left ventricle was greatly dilated and converted into fat, save its internal reticular part and papillary muscles.

The valves were all healthy, but "the aorta was studded with steatomatous and earthy concretions."* The remarkable change

* _Dublin Hospital Reports_, vol. ii., p. 216.

in the rhythm of respiration, of which this case presents the first example on record, was due, not to the condition of the heart, but to that of the aorta, as will appear in the sequel.

Doctor Gratiloup, of Bourdeaux, gives the case of a curate of that town, who died suddenly whilst in bed. On examination of the body, the heart was found greatly loaded with fat, and death had resulted from rupture of the right auricle.[*]

Mr. Adams is the next writer who illustrated this subject by cases and remarks, which, studied even in the light of present knowledge, will be admitted by all candid readers to possess extraordinary merit for originality and accuracy.[†]

All Mr. Adams' cases, like those hitherto noticed, are examples of fatty growth.[‡] He describes accumulated fat on the heart, a thin layer of muscle only remaining internally, and expresses the opinion that death in such cases may be sudden, and preceded by the usual symptoms of apoplexy. The two following cases are examples:

A gentleman sixty-eight years of age, had, in the course of seven years, no less than twenty pseudo-apoplectic fits, in which the breathing was stertorous and irregular, and the pulse slow; but no paralysis had succeeded. He was usually heavy and lethargic, and the memory failed at the approach of these fits. At the autopsy, both ventricles were found laden with fat, muscular tissue to the depth of a line only remaining on the internal surface.

A physician, aged sixty-eight years, who had had fainting fits with momentary loss of consciousness several times in the course of the last ten years of his life, was suddenly seized with pain in the chest, vertigo, and dyspnœa, on making an unusual effort with his arms lifted above his head. For six weeks, during which

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[*] *Archives Générales de Médecine*, 1822.

[†] *Dublin Hospital Reports*, vol. iv., 1827.

[‡] Doctor Ormerod has stated incorrectly, that the cases published in Dublin, prior to 1849, were examples of fatty degeneration, which he designates as "the Dublin form" of fatty heart. I have carefully looked into all the cases recorded in Dublin, and, with the exception of those published by Drs. Townsend and Stokes, I have failed to discover amongst them, previous to 1849, any examples of *fatty degeneration*, entitled to be so regarded from the description of them given by their authors. Of *fatty growth*, the cases published in Dublin before that date are numerous; and since then, both forms have been amply illustrated by the Dublin School.

he lived subsequently, there was no pulse to be felt in any of the arteries of his body, and no cardiac impulse beyond slight fluttering. He died worn out, and in a state of coma. The heart was found large, and loaded with fat; the aortic valves were completely calcified, and the first inch of the coronary arteries was calcified and entirely closed.

Doctor Townsend published[*] two cases of sudden death by rupture of the left ventricle in aged subjects. The walls of the left ventricle were "pale, soft, and flabby," and, in the seat of rupture, in an advanced state of fatty degeneration. At the base of the heart, and in the sulci, there was a thick layer of fat; and the walls of the right ventricle, which was more than half an inch thick, was composed of fat, with a layer of muscle, of only the thickness of paper, on its internal surface. In both these cases the coronary arteries were extensively calcified and partially closed. To this, as the cause of imperfect nutrition of the heart, Dr. Townsend attributes the fatty change which the organ had undergone. He likewise adverts to the absence of ulceration in the seat of rupture, and dwells upon the adequacy of fatty deposition and softening of the walls of the heart, to account for this accident.

Doctor C. J. B. Williams alludes to the subject of fatty softening of the heart.[†]

Professor R. W. Smith was the next contributor to the elucidation of this subject. He published two remarkable cases, in which death occurred so soon after admittance to hospital, that no opportunity was presented for noting symptoms. In both cases the heart was laden with adeps, *and free oil was found in the blood.* He expressed the opinion, since confirmed by the highest authorities in pathology, that the free oil found in the blood in these cases was of "constitutional origin," and due to imperfect assimilation, being an excess of the fat normally found in the blood.[‡]

Doctor Latham, in 1839-40, recorded four cases, all of which were good examples of fatty accumulation and fatty degenera-

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* *Dublin Journal of Medical and Chemical Science*, 1832, vol. i., p. 165.
† *Pathology and Diagnosis of Diseases of the Chest*, third edition, 1835, p. 191.
‡ *Dublin Journal*, vol. ix., July 1st, 1836.

tion combined. All four patients were males, and over sixty years of age, and two at least of the four were gouty. Two of the patients were subject to fainting fits; and in these, circumscribed aneurism of the left ventricle existed in conjunction with a fatty state of the heart, but rupture of the heart had not taken place, and death occurred by syncope. In the two remaining cases, rupture of the *septum ventriculorum* was the immediate cause of death; one of the patients surviving the occurrence of this accident eighteen hours, and the other three days, as judged by the final symptoms. Both these patients had had one or more attacks, resembling *angina pectoris*, in their last illness.

Doctor Latham held that a fatty state of the heart, however advanced, did not admit clinically of positive diagnosis; its symptoms and signs warranted only " a probable conjecture;" and even these were due, not to the structural alteration in the heart, but to the associated condition of dilatation which usually supervenes at an advanced stage of the affection.*

Four years later, Mr. Carmichael published an example of both forms of fatty change of the heart combined in the same individual. A clergyman, aged upwards of sixty years, temperate, but for a long time subject to palpitation and fainting fits, died suddenly, with symptoms of apoplexy, whilst at a wedding breakfast.

On the day succeeding that on which he died, the superficial veins of the forehead became turgid with air, and crepitant. The heart was covered with fat; the walls of the right ventricle, reduced to two lines in thickness, were almost entirely composed of fat, the remaining muscular tissue being greasy and readily broken down. The left cavities presented a similar condition, but in a less degree.†

In the same session (1840-41), Mr. Fleming communicated to the Pathological Society the particulars of a case of a similar character, but more valuable, because of the history of the patient's last illness, extending over several months, which he possessed. A gentleman, aged forty-five years, was rather sud-

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* *Lectures*, vol. ii., lecture xxvi.
† *Proceedings of the Pathological Society of Dublin*, Session 1840-41.

denly seized with nocturnal dyspnœa and palpitation, and shortly afterwards with loss of consciousness and hemiplegia. This latter attack lasted five hours, and during its continuance the pulse was nearly imperceptible. Under the use of a mustard foot-bath, a sinapism to the nape, and the administration of camphor and ammonia, recovery took place. There were four similar attacks between March and November, all of which were attended with jaundice, and were successfully treated in the same way The heart's action was strong, and accompanied with *bruit de soufflet;* the radial pulse was small and feeble, and occasionally imperceptible. The application of even a single leech caused alarming weakness, and there was extreme intolerance of opium; death was caused by another disease. The mitral valve was thick and incompetent, the left auriculo-ventricular opening very large, and there was excentric hypertrophy of the left ventricle The lungs were congested, partially emphysematous, and contained some apoplectic nodules. The state of the substance of the heart was not mentioned; but, from the symptoms, I think it may be regarded as fatty.

Doctor Stokes recorded the particulars of a case of sudden death by syncope in a man aged fifty years, with haggard expression of face, weak, but regular pulse, and feeble action of the heart, the sounds being faint, but distinct. The right ventricle was found loaded with fat, the left ventricle soft and flabby, and the valves all sound.[*]

In 1846 the following case was likewise noted by the same observer. A man aged fifty years was admitted into the Meath Hospital. He was very weak. The pulse varied from 35 to 40, but was regular. A systolic murmur existed in the ascending aorta, loudest at the right second costal cartilage. Death was sudden. The heart was soft and flabby, and covered by a thick layer of fat. The aortic valves were diseased, and the orifice narrowed. The aorta was atheromatous.[†]

Vicq-d'Azyr,[‡] and Partridge[§], in 1847, published examples

[*] *Proceedings of Pathological Society,* November 25th, 1843; *Dublin Journal,* vol. i., new series, p. 491.

[†] *The Diseases of the Heart and Aorta,* p. 312.

[‡] *Œuvres par Moreau,* tom. v., p. 365.

[§] *Medical Gazette,* 1847, p. 944.

of fatty degeneration of the voluntary muscles from paralysis; and in the same year Mr. Paget's important lectures, illustrative of the same subject, appeared.*

Doctor Bellingham exhibited before the Surgical Society of Dublin an example of fatty accumulation on the heart, especially on the right ventricle, with double aneurism of the arch of the aorta, taken from the body of a man aged twenty-seven, who died suddenly in a state of syncope. During life this man had exhibited no symptoms indicating a fatty condition of the heart.†

In the same year, and before the same Society, Dr. Henry Kennedy read a valuable paper on the subject of fatty disease of the heart; but chiefly in advocacy of the treatment of that disease by bodily exercise. He gave the particulars of two cases, which seemed to be examples of the compound form of the affection; that is, of fatty accumulation and fatty degeneration combined. In both cases the patients were of advanced age, and subject to fainting fits without loss of consciousness.‡

In the discussion of Dr. Kennedy's paper, Professor Harrison, while dissenting from the plan of treatment recommended, exemplified the danger of exercise in fatty disease of the heart, by reference to the case of an old gentleman who, whilst making a journey in his carriage, suddenly died in consequence of a jolt of the vehicle. The heart was found laden with fat, and its substance in a state of fatty degeneration.

Doctor Latham Ormerod in this year (1849) formally recognized two distinct varieties of fatty change in the heart; viz., fatty accumulation on the heart in fat persons, and fatty degeneration of its substance.§ The latter form was that which was especially treated of in his memoir. This he describes as exhibited in fawn-coloured patches on the inner or outer surface of the heart; and, microscopically examined, these patches are seen to exhibit the following changes. The fibres immediately adjoining show interruption in the continuity of the transverse striæ, which now appear as transverse lines of dark dots; more

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* *London Medical Gazette,* 1847, lectures v. and vi.
† *Dublin Medical Press,* vol. xxi., April, 1849.
‡ *Ibid.,* vol. xxii., December, 1849.
§ *London Medical Gazette,* vol. ix., p. 739.

internally the intervals between the dots are longer, and the latter assume a linear arrangement in the length of the fibre; and still nearer the centre they are scattered irregularly, constituting the "granular condition," or granular stage. Towards the centre of these patches oil dots appear at distant intervals, and finally they take the place of the dark granules within the sarcolemma; this is the "fatty stage." The condition just described he declares he has never met with in the auricles. He denies chemical "conversion" into fat; but admits a "substitution" of fatty for the disintegrated muscle-particles in the process of nutrition.

In 1853, Paget's great work appeared, in which, amongst many subjects, that of fatty changes in the heart is fully considered.[*]

In 1854, Dr. Stokes' valuable clinical treatise[+] was published; in it fatty change of the heart in its two forms is amply discussed and illustrated. Since that date the contributions to the pathology and the clinical history of fatty disease of the heart have been very numerous, and the affection may be considered to have taken a recognized position in nosology.

The two forms of fatty change of the heart are usually, but not always associated, and the apparently opposite circumstances under which they are met with may serve to explain both the rule and the exceptions.

Fatty growth upon the heart is most frequently associated with general obesity, and occurs at that period of life, and in that class of society, in which deposition of fat is of ordinary occurrence in the natural or physiological order. But beyond a certain limit, obesity is not compatible with the active nutrition, or the functional vigour of muscle, because of the impediment to muscular exercise, and the consequent impairment of muscular nutrition, which it involves. In many instances, also, it is associated with the gouty diathesis and the attendant atheromatous change in the arteries, by which the nutrition of tissue is directly interfered with through an inadequate supply of blood.

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[*] *Lectures on Surgical Pathology,* vol. i., p. 93, *et sequent.* In some valuable lectures published in the *London Medical Gazette* several years previously, Mr Paget had fully recognized the distinction between fatty growth and fatty metamorphosis.

[+] *The Diseases of the Heart and Aorta.*

In many cases, likewise, by the mere weight of incumbent fat, muscle undergoes absorption and atrophy; and where, as most frequently happens, these concomitant changes are met with in the heart, even the residue of muscular substance is unsound. Nevertheless the portion of muscular fibre as yet unaffected by the pressure of the adipose mass, may, if uninfluenced by the other causes of malnutrition previously mentioned, be actually found in a state of textural integrity. This condition is, however, strictly exceptional, and a good illustration of it is presented in the subjoined engraving (Fig. XXXIV.) from the heart

FIG. XXXIV.
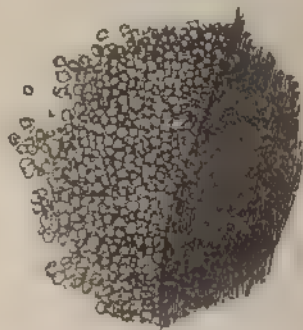
Fat cells and muscular fibre ; magnified 24 diameters.   Mr. B.

of Mr. B. (*vide* Aneurism). It will be observed that whilst the accumulated fat has caused, by its pressure, all but complete absorption of the walls of the right ventricle, or at least, has replaced the muscular substance of the walls nearly to their entire depth, the thin muscular layer which remains is histologically sound. It is further noticeable that the line of union between the adipose substance and the muscular structure of the heart is quite abrupt, and that no fatty infiltration of the latter has taken place. In Fig XXXV. the adipose tissue from the surface of the same heart is represented still more highly magnified ; and in the next engraving (Fig. XXXVI.) the unaltered muscular fibre, equally magnified, is represented.
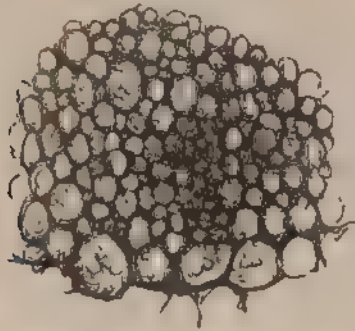
Fig. XXXV.

Fat cells ; × 210 diameters. Mr. B

Fig. XXXVI.

Unaltered muscular fibre ; × 210 diameters. Mr. B

In this case there were present none of the ordinary symptoms of fatty disease of the heart, except the comparatively insignificant one of weak pulse; and no physical sign indicative of that condition, with the single exception of a feeble first sound. Death took place by rupture of a large aneurism of the aorta into the pericardium.

Judging from the experience afforded by this single example of fatty growth upon the heart, causing absorption in great part of its proper structure, but unattended with fatty infiltration or fatty degeneration of that portion of the muscular wall which remained (and it is the only one of the kind which has come

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under my notice), I would say that simple accumulation of fat upon the heart is, *per se*, unannounced by evidence, either general or special, sufficient to warrant a positive diagnosis.   In all the examples of sudden death by simple syncope, or by rupture of the heart, associated with epicardial growth of fat, which I have met with or seen recorded, there was reason to conclude, where a positive statement to that effect was not made, that the muscular substance of the heart had undergone, in a greater or less degree, the change of fatty degeneration.   Thus, it will be found that the muscular structure of the organ was represented as being fawn-coloured, easily torn, or soft and flabby.   Any one of these statements would justify the suspicion of fatty change of the muscular fibres.

Fatty growth upon the heart is usually traceable to causes identical with those which give rise to general obesity; especially to sedentary habits, and indulgence in spirituous liquors.

True *fatty degeneration*, or transformation of the muscular fibres of the heart, is a much more serious affection, and differs from the preceding genetically as well as histologically.

The pathology of this form of the affection, quite irrespectively of the estimate which may be formed by individuals of the value of pathological doctrines generally, is of undoubted importance in regard to its probable issue and treatment.

Doctor Latham Ormerod maintained that fatty degeneration is the product of a veritable perversion of nutrition, attended with sub-oxidation of the food and of the tissues, and resulting in the deposition of morbid fat or olein.[*]   He denied the identity of adipocire with the product of fatty degeneration.

This is likewise the opinion of M. Ch. Robin, who designates the change as " fatty substitution."[†]

Mr. Paget, on the contrary, is of opinion that it results from a *defect* of nutrition, and consequent chemical changes of tissue of an atrophic character.[‡]

Rindfleisch, as already stated, holds the same opinion.

Doctor Quain regards the change as physical or chemical, and

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[*] *St. Bartholomew's Hospital Reports*, vol. iv., 1865.

[†] *Chimie Anatomique ;* see Flint, *Diseases of the Heart*, second edition, 1870, p. 97.

[‡] *Lectures on Surgical Pathology*, 1853, vol. i., p. 93.

analogous to the decay of dead structures outside the body. It is, therefore, in his opinion, closely allied to, if not identical with, adipocirous transformation.*

I believe that fatty degeneration of muscle is a veritable necrobiosis, differing only in degree from the changes which take place when the controlling influence of tissue-vitality has been entirely withdrawn. In the former case, the process of tissue-death is slow and progressive, under the modifying influence of a feeble and inadequate nutrition. In the latter, nutrition being entirely and finally suspended, it is rapid and complete.

The circumstances which so impair the nutrition of the heart, as to induce fatty transformation of its substance, may be local or general. In the former case the transit of nutrient material is mechanically obstructed, as by calcification or occlusion of the coronary arteries, or some of their branches. Thus a larger or smaller district of muscle, according to the extent of arterial disease, or the size of the vessel affected, may be partially or wholly deprived of pabulum, and, under the influence of vital or physical agency, assume new forms according to its chemical affinities.

Most authorities are agreed that a similar series of changes may be induced by antecedent inflammation of the substance of the heart. Hence the gravity of myocarditis. Bouillaud, Hope, Virchow, and Rindfleisch advocate this view.†

Virchow maintains that dilatation of the heart, as implying antecedent irritation or sub-inflammation of its substance, has a similar effect.

The constitutional causes of fatty degeneration of the heart are such as interfere with general nutrition. Amongst these may be mentioned, long continued inadequate supply of food, especially if associated with severe physical labour, recurrent hæmorrhage, and tubercular or cancerous marasmus.

Drs. Ormerod and Quain call special attention to the frequent association of fatty degeneration of the heart with habitual loss of blood. Of this I have noted several examples.

* *Medico-Chirurgical Transactions*, vol. xxxiii., 1850

† Wagner, on the authority of Reynaud, states that fatty degeneration followed pericarditis in seventeen out of thirty five cases in his practice. (See Flint, *Diseases of the Heart*, 1870, p. 98).

The above mentioned general causes, having reference to insufficient nutriment and excessive labour of body, apply especially to the poor and working classes. On the other hand, those of a local character, involving atheromatous or calcareous transformation of the coronary arteries, and inflammatory softening of the heart, are found to operate in about equal proportion upon the rich and gouty, and the poor and ill fed members of the community.

Thus, whilst excessive deposit of fat upon the heart is the almost exclusive appanage of the rich, fatty degeneration of its substance without excessive superficial deposit, is apportioned about equally between the rich and the poor. When it is remembered that, in the great majority of examples of fatty growth upon the heart, fatty degeneration of its muscular structure likewise exists, it will be conceded that the wealthy classes of society enjoy an unenviable preeminence in regard to liability to fatty disease of the heart.

This conclusion is not quite in accord with that at which Quain has arrived. Of forty-eight cases of both forms of fatty change of the heart, respecting which he has noted the class of society to which the patient had belonged, sixteen were of the upper, fourteen of the middle, and eighteen of the lower class.

The process of fatty transformation of the heart may be conveniently divided into three stages, as determined by microscopical inspection : namely, the stage of *granular transformation* of the fibres, that of their *substitution by oil drops*, and the stage of their *obsolescence*. Dr. Ormerod maintains that the granular is distinct from the fatty change, and involves no increase of oil in the muscular substance. In this opinion I cannot agree with him, and I find myself rather in accord upon this point with Drs. Quekett and Quain, who regard the granular transformation of the muscular fibres as representing the first stage of their conversion into fat.*

In this stage, the muscular fibres are well defined in outline, and present transverse striation with the usual distinctness over a greater or less extent of the field of vision, but inversely as the progress in histolytic change actually made. Over a variable portion of the field, likewise, and directly as the advance of fatty

* *Lectures on Histology,* p. 196, and *loco citat.*

change, the transverse markings of the fibres are interrupted, and represented by lines of dark molecules at right angles with the axis of the fibres. In certain parts of the same section, and in-dicating a more advanced degree of change, these transverse lines of dark molecules are replaced by longitudinal rows of similar bodies; this is apparently due to the twofold cause of de-rangement of mutual apposition of the fibres, and conversion of entire fibrils or strings of sarcous elements into minute oil dots. The condition indicated, under the various transitional changes just mentioned, is represented in a preceding engraving. Fig. XXXI., p. 553, shows the granules at a very early period, when the parallelism of the transverse series is scarcely deranged; and Fig. XXXVII. (Mary A.) subjoined, shows the granular transfor-mation in a still more advanced stage.

Fig. XXXVII.
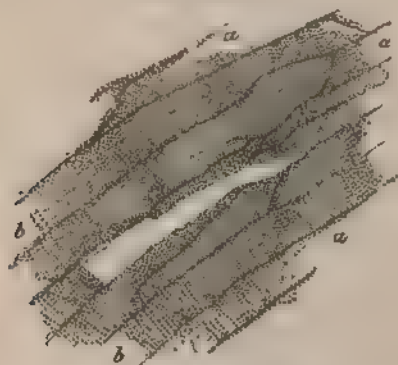
Granular degeneration.
(b) Early stage; disintegration of transverse striæ into granules.
(a) A more advanced stage; longitudinal arrangement of granules.
× 222 D. Case 61 (Mary A.).

In the further progress of change, the minute dark granules coalesce and form larger spherules or oil dots, distinguishable by a luminous centre and a dark outline. These differ considerably in size, and are disposed at unequal intervals in the length of the fibre. They are usually crowded around the nucleus, as noticed by Paget and Rindfleisch. The latter assumes, but in my opinion incorrectly, that the appearance is due to the diffusion

of oil amongst the fibrils, and its accumulation in the interstices
formed around the nucleus by their divarication.   It is not diffi-
cult to satisfy one's self that the molecules of oil, when thus aggre-
gated, are still within the limits of the fibrillary walls, and though
grouped around the nucleus, preserve the linear arrangement of
the fibrils. This crowding of the oil dots within their respective
fibrils, I incline to attribute rather to the vital or nutritive at-
traction of the nucleus than to a simple mechanical agency.   In
a more advanced state of transformation, what may be desig-
nated the third stage, or that of obsolescence after Ormerod,
the fibrils would seem to have undergone solution, and the
contained oil to have become diffused, forming large oil drops
within the sarcolemma. All appearance of definite structure
in the fibre is now utterly effaced; large portions of it present
a perfect blank, and in these situations, as determined by its
still well defined outline, it is reduced in diameter, whilst it is
enlarged or bulged out in those portions where the oil drops
have congregated.   It rarely happens that this advanced degree
of change has extended beyond a very limited area, because its
further progress is usually interrupted by death, arising from
rupture of the heart where it has been attained, or from syncope
without apparent disintegration of structure.   The accompany-
ing engravings (Figs. XXXVIII. and XXXIX., John McD.)
show the several anatomical peculiarities of the second and
third stages just described.   Where fatty degeneration is due
to obstruction of minute branches of the coronary arteries,
its area is often very limited.   It not unfrequently also ap-

FIG. XXXVIII.
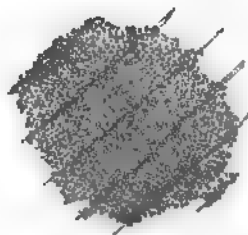
John McD.   × 222 diameters.
Fatty degeneration (early stage).   See Case 57.

Fig. XXXIX.

John McD.

(1) Transverse section of fibre; sarcolemma visible and partially collapsed, with numerous escaped oil dots.

(2) Transverse section of fibre, sarcolemma not visible.

(3) A fragment of a fibre seen in length, showing partial obsolescence. The oil drops are collected in groups in the situation of the nuclei; but most of them have escaped from the sarcolemma. See Case 57.

pears upon the inner or outer surface of the heart, in the form of light fawn-coloured spots, varying in size from the diameter of a pin's head to that of a split pea. This appearance is most frequently observed on the papillary muscles and fleshy columns, and is most probably due to antecedent endocarditis or pericarditis, as the neighbouring serous surface is generally opaque, and the endothelium thickened. In either case, these limited areas present, as it were in concentric circles, the different stages of fatty transformation above described; the outer zone exhibiting the earliest condition of granular change, and the centre that of complete abolition of structure, as noticed by Ormerod. Whilst, in the majority of cases, superficial accumulation of fat upon the heart coexists with fatty degeneration of its substance, interstitial or interfibrous deposition of fat is very rarely met with in conjunction with fatty degeneration; unless, as remarked by Sir J. Paget, the oil which has escaped by disintegration of the sarcolemma, be so regarded.

The *period of life* at which fatty degeneration most frequently occurs, though advanced, is not absolutely limited. From Tables V., VI., and VII.,* comprising a total of ninety-five cases of fatty degeneration of the heart collected from three sources; namely, the Transactions of the Pathological Society of London since

* Vide p. 648, et seq.

1850, those of the Pathological Society of Dublin subsequent to the same date,* and my own private records, I find the following results in regard to the ages of the subjects of this affection, given in decennial periods.

		Cases.	
Under 20 years	London Pathological Society ...	6	
	Dublin Pathological Society ...	2	= 9
	The Author's returns	1	
20 to 30 years	London Pathological Society ...	6	
	Dublin Pathological Society ...	2	= 9
	The Author's returns	1	
30 to 40 years	London Pathological Society ...	5	
	Dublin Pathological Society ...	0	= 6
	The Author's returns	1	
40 to 50 years	London Pathological Society ...	10	
	Dublin Pathological Society ...	2	= 22
	The Author's returns	10	
50 to 60 years	London Pathological Society ...	4	
	Dublin Pathological Society ...	2	= 8
	The Author's returns	2	
60 to 70 years	London Pathological Society ...	13	
	Dublin Pathological Society ...	9	= 27
	The Author's returns	5	
70 to 80 years	London Pathological Society ...	6	
	Dublin Pathological Society ...	0	= 7
	The Author's returns	1	

$$\overline{88}$$

From these combined returns, it would appear that fatty degeneration of the heart is most prevalent between the ages of sixty and seventy years; and next, between forty and fifty. The youngest patient was five, and the oldest seventy-nine years. My own returns show it to be most common from forty to fifty years, and *twice* more frequent within that decennial period than

* As Doctor Quain's statistics (*Medico-Chirurgical Transactions*, vol. xxxiii.) were compiled in 1850, and contain in abstract all the cases previously published, I deemed it most convenient, and possibly most conducive to the interests of medical science, to take this date as my starting point, with the view of presenting one unbroken series up to the present time. With that object in view, I have, as far as was consistent with fulness of detail, assimilated my own returns, and those of the Dublin Pathological Society, to the Tables of Drs. Ormerod and Quain.

between the ages of sixty and seventy. I believe, indeed, that the disease under consideration belongs especially to the middle period of life, from forty to sixty years; 40·5 is the average given by Ormerod.

With regard to *sex*, Bizot maintains that fatty degeneration of the heart is more common in women than in men.* Herein he is undoubtedly in error, as the following summary will show. Of a total of 55 cases collected from the London Pathological Society's Transactions, 35 were males and 20 females. A total of 18, obtained from the Transactions of the Dublin Pathological Society, furnishes 12 males and 6 females; and my own 22 cases yield 17 males and 5 females. Thus, out of a total of 95 cases obtained from these three sources, 64 were males, and only 31, *or less than one-half*, were females. Of Ormerod's 25 cases, 18 were males and 7 females; and Quain found the male subjects of the disease more numerous than the females, by 4 to 1.

The general condition of the patients may vary considerably; 9 of Ormerod's were fat, 6 were thin, and 2 were pale. Amongst his 25 cases there were 10 inebriates.

The *duration of illness*, from the date of the earliest symptoms connected with the disease to its fatal termination, varied in Quain's cases from a *minimum* of two to a *maximum* of 16 years. In Table V. (London Pathological Society), it varied from 4 days to 8 years. In Table VI. (Dublin Pathological Society), the extremes were 8 months, and 40 years; and in my cases, 3 weeks, and 16 years. Ormerod traced the origin of the disease to hæmorrhage in 4 cases, to phthisis in 3, and to anasarca in 10 cases. It is manifestly impossible to fix, even approximately, the date of commencement of structural alteration of the heart. It is only when this has made some, and even considerable progress, that it is declared by symptoms sufficiently pronounced to arrest the patient's attention. Thus, in a mere historic sketch of the case, the duration of ill health is usually under-estimated. Patients have repeatedly informed me that the first intimation which they had of anything serious being amiss with them, consisted in the sudden accession of violent pain in

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* *Memoires de la Société d'Observation*, tom. i., p. 351.

the region of the heart or epigastrium, with palpitation and dyspnœa on making some unusual physical effort, or on hearing some startling news. After this event they have never been again in their accustomed health (see Cases 23 and 51). In such cases a heart already far advanced in disease, but hitherto competent to discharge the routine duties of a quiet existence, has been surprised and overwhelmed by the sudden imposition of an extraordinary task, and never again been able to recover its dynamic equilibrium.

Again, in a case where structural disease of the heart had never been suspected, so robust had the health apparently been, the first announcement of its existence may consist in the rapid sinking of the patient, under the shock of an otherwise trivial acute disease. In this way, a mild attack of influenza, of bronchitis, of rheumatic or essential fever, may prove unexpectedly and rapidly fatal.

Mr. Paget truly remarks: " They who labour under it (fatty degeneration of the heart) may be fit for all the ordinary events of a calm and quiet life, but they are unable to resist the storm of a sickness, an accident, or an operation."*

Where a person, the subject of fatty degeneration of the heart, is attacked with acute disease, the pulse becomes very rapid, as truly remarked by Dr. H. Kennedy. Hence the evil augury of very rapid pulse in febrile disease.

I have already intimated that fatty degeneration of the heart is not unfrequently secondary to hypertrophy, whether the latter affection be the result of valvular lesion, atheroma of the arteries, or Bright's disease.

Doctor T. K. Chambers found the heart enlarged in 29 out of 49 cases of fatty degeneration. And in 12 out of 35 cases of hypertrophy of the heart, secondary to renal disease, Wagner discovered fatty degeneration of its substance.† In 31 out of the 95 cases summarised in my Tables (V. VI. and VII.‡), hypertrophy existed in conjunction with fatty degeneration of the heart; that is, in 13 out of 55 in Table V.; in 4 out of 18 in

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* *Lectures on Surgical Pathology,* 1853, vol. i., p. 129.
† Flint, *Opus citat.*
‡ *Vide* p. 648, *et seq.*

Table VI.; and in 14 out of 22 in Table VII. This is, no doubt, most commonly the immediate cause of the fatal symptoms, when death proceeds from the heart, in cases of hypertrophy from renal or valvular disease, or from atheroma of the aorta. Where failure of the heart in a gouty subject, as indicated by irregular action and palpitation, quickly followed by paroxysmal dyspnœa and dropsical effusion, becomes the signal of a "break up" of the constitution, as not unfrequently happens, fatty degeneration and dilatation of the heart will be found the last links in the chain, of which atheromatous change of the aorta, hypertrophy of the heart, and granular degeneration of the kidneys constitute the preceding links, and inversely as the order in which they have been just mentioned.

That fatty degeneration of the heart may arise, as has been alleged, from the tuberculous or cancerous cachexia; from pyæmia or septicæmia; or as a result of any of the essential fevers,[*] irrespectively of inflammatory complications of the heart, is, I think, more than doubtful. I believe, however, that slow poisoning by phosphorus or arsenic may give rise to it, although I have not myself met with an instance of the kind.

The *symptoms* indicative of a fatty condition of the heart are individually of little value; but combined in groups, as will be presently shown, they assume an affirmative significance.

I have already intimated that mere fatty growth upon the heart, without degeneration of its muscular substance, is not announced by any special symptoms or signs, save in so far as it may impede the motions of the heart mechanically, or cause thinning of its walls by continuous pressure upon them.

The symptoms regarded as proper to fatty degeneration of the heart have reference to the organs of the circulation, the brain, and the lungs; and, in a subordinate degree, also, the kidneys and the liver.

There is, in all cases, evidence of partial failure of the circulation, under the form of weak, irregular, intermittent, or very slow action of the heart and radial pulse; precordial oppression

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[*] Doctor Murchison, in the first edition of his great work, *A Treatise on Continued Fevers*, 1862, declared typhous softening to be identical with fatty degeneration of the heart.

or pain of an intermittent character, the latter frequently extending down the left arm as far as the elbow; palpitation on making any unusual effort, physical or mental; inability to resist the operation of heat or cold, or preserve the thermal equilibrium under extremes of temperature; pallor of surface; readiness to perspire; and recurrent syncope.

These symptoms of a feeble circulation were present in greater or less number in 42 out of the 95 cases represented in these Tables; that is, in 15 of Table V.; 12 of Table VI.; and 16 of Table VII. They were exhibited in 35 out of 108 cases given by Ormerod and Quain. They are rarely exhibited in the aggregate in any case of fatty transformation of the heart; nor when present as ordinary features, are they constant in any given case. The most frequently present and the most constant, are weakness or inability to endure fatigue, feeble pulse, palpitation and precordial oppression, accompanied by dizziness and a tendency to syncope after an effort of any kind, and want of the power of thermal adjustment.

The symptoms enumerated by Beau, Reynaud, and, more recently, by Seitz,* as indicative of " strained heart " (cœur forcé, Beau); viz., breathlessness and palpitation, accompanied by a feeling of weight and oppression at the heart after trivial exertion; and, after a more severe effort, pain and a sense of constriction in the region of the heart, followed by chill, cough, and hæmoptysis, and occasionally by syncope, deserve special notice. The symptoms assume a more urgent character in the progress of the case, and are accompanied by a feeling of impending suffocation. Venous congestion, œdema, passive engorgement of the liver, and effusion into the serous cavities, follow in succession. The pulse is irregular and feeble. Precordial dulness may be either normal or extended ; but the impulse of the heart is weak, and its sounds are dull and ill pronounced. Usually there is no cardiac murmur; but occasionally a faint murmur is heard with the first sound, or an indistinct frottement. Death may take place suddenly, or by a slow process of asphyxia.

* Deutsches Archivs f. Klinische Medicin, vols. xi. et xii.

On examination of the body after death, no evidence of organic disease of the heart, with the exception of a few muscular fibres, "here and there," in process of fatty degeneration, can be discovered.

The history above given, and the symptoms enumerated, are certainly those of fatty disease of the heart; and in every case in which they have been presented to me, the heart, when examined after death, has been found in a more or less advanced stage of fatty change.

Irregularity of the pulse is somewhat more common than intermittence, and both are inconstant, and associated with temporary exhaustion. Irregularity of pulse was present in 5, and intermittence in only 1 out of 95 cases. The triple combination of pain in the region of the heart, dyspnœa, and syncope, constituting angina pectoris, is still less frequent. It was exhibited in only 5 out of 83 cases given in Dr. Quain's Tables, and in 8 out of the 95 given in mine.

Slow pulse, that is, under 60 in the minute, is very rare. I have not had a single example amongst the cases recorded in this chapter, and have met with only one in my experience. A man aged seventy-five years, with œdema of the feet and other symptoms of weak heart, exhibited a pulse of only 30 in the minute in the recumbent posture, but perfectly regular. The sounds of the heart were somewhat dull, but free from murmur.[*]

Heberden mentions two examples of slow pulse.[†] A gentleman of eighty years, not ill, had a pulse which was occasionally but 26 in the minute, seldom above 30, and once only to his knowledge amounted to 42. He saw another person whose pulse at the beginning of illness was represented as only 12 to 16. But, in reference to such examples of extreme slowness of pulse, he judiciously remarks, that the radial pulsations do not represent fully those of the heart.

Doctor James Johnston gives the case of a gentleman, aged seventy-one years, whose pulse was very slow, and who after-

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[*] *Vide* "Rhythm of the Heart's Action," by the Author, *Dublin Quarterly Journal of Medicine*, August, 1865.

[†] *Medical Transactions of the London College of Physicians*, 1786, vol. ii., p. 18.

wards died suddenly. The heart was soft and brittle, but the great vessels were healthy.*

Doctor Spens reports the case of a mechanic, aged fifty-four years, subject to headache and to attacks of syncope and convulsions, whose pulse was ordinarily from 23 to 26, and some hours before death, which took place suddenly, fell to 10 and 9 in the minute. The brain was found soft and anæmic; there was a good deal of serum in the ventricles and meshes of the pia mater. There is no notice of the state of the heart.†

A gentleman of sixty-four years had fainting fits, followed by slow pulse, not exceeding 24. On one occasion it fell to 10, 9, 8, and 7½, as registered by Dr. Mitchell and Mr. Cullen. Failure of the pulse preceded the attacks of syncope, which were attended with convulsive twitchings of the face, and foaming at the mouth. Autopsy by Mr. Liston : left ventricle thin, and both auriculo-ventricular openings large; the lining of both ventricles was thick and opaque.‡

Rouchoux mentions that the First Napoleon's pulse was habitually only 40, and weak.§

Mr. B. W. Richardson reports ‖ the case of a gentleman, aged seventy-eight years, who was also seen by Mr. Adams and Dr. Stokes, whose pulse was only 38, and afterwards fell to 24. He had had repeated attacks of syncope with dyspnœa. The feet were swollen; there was no *arcus senilis*. On examination of the body, the heart was found large and flabby. There was a large deposit of fat upon its surface, and its substance was in a state of fatty degeneration. The aorta and the coronary arteries were atheromatous; the brain was not examined.

Mr. Richardson reports another case, in which the pulse varied from 32 to 40. The patient, who was aged seventy-three years, had been subject to dyspnœa and fainting fits, not followed by paralysis. There was œdema of the feet, albuminuria,

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* *Memoirs of the Medical Society of London*, 1787, vol. i., p. 376. See also Mr. B. W. Richardson, *Dublin Journal of Medicine*, vol. xii., 1852.

† *Medical Commentaries*, 1792, *loco citat.*

‡ *Medico-Chirurgical Transactions*, vol. xxiv., p. 73. See Dr. Richardson, *loco citat.*

§ *Dictionnaire de Médecine*, "Pouls."

‖ *Loco citat.*

visible pulsation of the arteries, and *arcus senilis*. There was likewise a systolic murmur both at the apex and base of the heart. Death was caused by effusion into the serous cavities. The heart was found large and soft, exhibiting both fatty growth and degeneration. The aorta and its principal branches, and the coronary arteries, were atheromatous The mitral and the aortic valves were calcified, rough, and incompetent.

Doctor Stokes gives a list of seven cases of permanently slow pulse observed by himself, or quoted from Dublin physicians. He draws special attention to the combination of slow pulse and a tendency to syncope, with disease at the orifice of the aorta. In reference to his cases he says : " In five, organic disease of the aorta, or the valves, or both, was discovered on dissection, and in four a manifest aortic murmur existed. In two of the cases the second sound was normal, and in two there was the murmur of regurgitation in the aortic valve."* Thus it would appear that, in the opinion of Dr. Stokes, the cause of slow pulse is to be sought primarily in the heart and aorta, and secondarily in the state of the cerebral circulation.

Doctor Law mentions† the case of a nobleman who had been subject to syncope, and whose ordinary pulse was only 25. On examination of the body, the arachnoid membrane was found opaque and thick, the pia mater infiltrated with serum, the convolutions of the left cerebral hemisphere had shrunk to half their normal size, and the cerebral substance, both grey and white, softened over one-third of the hemisphere, and nearly to the depth of the ventricles, which were full of serum The arteries at the base of the brain were atheromatous. The pericardium was partially adherent to the heart ; there were ossific plates in the mitral valve, which, however, was competent ; and the aortic valve was thickened, partially ossified, and incompetent.

Doctor Quain gives 8 examples of slow pulse amongst his cases. Of these, the slowest was 24 in the recumbent and 32 in the sitting posture.

Doctor Ormerod regards slow pulse as characteristic, in an

* *Dublin Quarterly Journal of Medical Science*, new series, vol. ii., August, 1846.
† *Dublin Journal*, vol. xvii., May, 1840.

especial manner, of fatty degeneration of the heart. In this opinion I am sure he is right; but the cause must be sought rather in the consequences of a weak left ventricle and atheromatous arteries, than in these conditions themselves; namely, in enfeebled innervation of the heart from defective nutrition of the cerebro-spinal centres and cardiac ganglia. It is worthy of remark that, in most of the reported cases of slow pulse in which a careful dissection has been made, the coronary arteries were extensively diseased, and the substance of the brain soft and anæmic; whilst on the other hand, either of these lesions singly may be commonly met with, unassociated with slow pulse.

Doctor Todd declares that hearts weak by anæmia, or fatty degeneration, may pulsate *more slowly* in the sitting or the standing, than in the recumbent posture of the body; and he expresses the opinion that this is due to the overloading of a weak heart in the erect posture. Moreover, he thinks that the equality of rate of pulsation in all postures of the body, noticed by Graves in hypertrophy with dilatation of the heart, was due to the same cause.* But, manifestly, if this were so, pulsation should in the latter, as in the former cases, be slower in the sitting than in the recumbent posture; I have never met with an instance of the kind mentioned by Dr. Todd.

Irregularity and intermittence of the action of the heart are kindred phenomena, and due to deranged rather than to imperfect innervation. They are not, however, like slow but regular action, pathognomonic of fatty transformation of the muscular fibres of the heart, for both are frequently met with where no such transformation, and, indeed, no tissue-change whatever of the heart exists. But, whilst irregularity of the arythmical or non-periodic kind is never unassociated with organic disease of the heart, simple intermittence very often, and indeed most frequently, is due to other causes; for example, general debility from any cause, syncope from sudden loss of blood, profound dejection, and certain chronic diseases of the brain. Irregularity in its typical form is, therefore, in general significance, a much more formidable sign than intermittence. Rhythmical irregularity, or simple derangement of *rate* in the heart and pulse is

* *London Medical Gazette*, 1851, vol. xlvii., lecture xx.